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ENVIRONMENTAL IMPACT STATEMENT

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Department of Health and Environmental Sciences
STATE OF MONTANA HELENA, MONTANA 59601

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January 3, 1979

TO: Interested Persons

This is the draft Environmental Impact Statement on the ambient standards recommended by the Montana Ambient Air Quality Standards study (MAAQS). Comments on this draft should be submitted to MAAQS at the Air Quality Bureau by 5:00 p.m. March 1, 1979. There will be no extension of this deadline.

The final Environmental Impact Statement is scheduled for issuance about the first of April. Following issuance of the final EIS, the Board of Health and Environmental Sciences will accept written testimony on the proposed standards for four weeks. After that, there will be a six-week period for the filing of rebuttal arguments to prefiled testimony. Following review of the written testimony and rebuttal, the Board may call witnesses to clarify disputed or unclear points.

The earliest the Board could act to adopt standards would be some time in September.

A detailed schedule of hearing dates and testimony deadlines will be provided with the final EIS.

Sincerely,

Michael D. Roach, Chief
Air Quality Bureau

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PREFACE

This environmental impact statement is intended to help the public decide what ambient air quality standards Montana should have. It is written in reasonably simple language so as to be understandable to persons without a scientific background. There are places where the scientific specifics may suffer from this simplification, and other places where such specifics were difficult to put into common terms.

We regret such shortcomings and invite the reader to point them out to us so that we may try to eliminate them prior to the issuance of the final environmental impact statement.

MONTANA AMBIENT AIR QUALITY STANDARDS STUDY
DRAFT ENVIRONMENTAL IMPACT STATEMENT

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SUMMARY

In the fall of 1977 the Montana Department of Health and Environmental Sciences (DHES) was considering enforcement action against some Montana industries for violations of the administrative regulation on Ambient Air Quality Standards (ARM 16-2.14(1)-S14140). This rule had been on the books more than ten years and had been regarded during that time as an enforceable regulation. During the research in preparation for the enforcement action, however, it was discovered that there was some doubt whether the Board of Health and Environmental Sciences adopted the standards with the intent that they be directly enforceable.

The confused status of the ambient air quality standards did not leave the state without regulations to control air pollution. Still in effect were the emission regulations, which stipulate what amounts of any given pollutant may be emitted by any given source. In many cases the emission regulations are sufficient to keep ambient concentrations below harmful levels under ordinary conditions. However, variable situations such as recurring periods of calm winds, the presence of other sources to increase the total emissions in a given area, or the presence of persons or parts of the environment particularly susceptible to air pollution all indicate the need for enforceable ambient standards to supplement the emission regulations. Ambient pollution concentrations cause whatever effects that are suffered by the public and property. If emission regulations for any reason fail to provide sufficient protection for air pollution under special situations, an enforceable ambient standard provides another instrument to protect the public health and environment.

When advised of the questionable enforceability of the Montana Ambient Air Quality Standards, the Board indicated it wanted the state to have enforceable standards. It was decided that before such standards were adopted anew, there should be a thorough review to determine whether the old standards were still appropriate or whether scientific research completed since their adoption indicated different standards were needed.

Subsequently, $147,000 was provided to hire a staff and consultants to review all the literature reasonably available and pertinent to air pollution in Montana. The literature reviewed pertained to pollutants already regulated by the old standards and emission rule and to several others, including the following: sulfur dioxide, total reactive sulfur (sulfation), acid mist, total suspended sulfate, respirable particulate, total suspended particulate, settleable particulate, lead, arsenic, cadmium, beryllium, carbon monoxide, nitrogen dioxide, photochemical oxidants, particulate and gaseous fluoride, hydrogen sulfide, and polycyclic organic matter. Standards for visibility and odors also were considered.

Following the review, it was concluded that ambient standards were feasible in Montana for the following pollutants: sulfur dioxide, total suspended particulate, settleable particulate, lead, carbon monoxide, nitrogen dioxide, photochemical oxidants, hydrogen sulfide and visibility. There was insufficient research and information to indicate a current need for ambient standards to control arsenic, cadmium, polycyclic organic matter, beryllium, reactive sulfur, respirable particles, suspended sulfate, sulfuric acid mist, or odors. Therefore, no standards are proposed for these. However, the Air Quality Bureau will continue to review new research results as they become available, and will recommend standards if new information indicates the need.

Nearly all the standards proposed in this impact statement are intended to keep concentration of the various pollutants at concentrations lower than those found to be associated with health effects in humans. The exception is hydrogen fluoride, which causes substantial economic damage to plants and animals at levels which apparently are much lower than those where humans are directly affected. The proposed fluoride standard therfore is based on the level of hydorgen fluoride known to cause such damage.

It is expected that the recommended standards for each of the pollutants will protect not only human health but, to a major extent, the environment.

Most of the standards include a "margin of saftey," meaning the concentration allowed by the standard is lower than the concentration known to actually cause an effect.

The proposed standards are as follows:

Sulfur dioxide:

0.02 ppm	Annual average not to be exceeded
0.10 ppm	24-hour average not to be exceeded more than once a year;
0.40 ppm	Hourly average not to be exceeded more than once a year

Particulate matter:

75 ug/m^3	Annual average, not to be exceeded
200 ug/m^3	24-hour average, not to be exceeded more than once a year;

Settled Particulate matter:

10 gm/m^3	30 day average

Visibility:	Particle scattering coefficient of 2×10^{-5} per meter, not to be exceeded more than once a year; as measured by the integrating nephelometer.

Fluoride:

1.0 ppb	24-hour average
0.30 ppb	30 day average
0.13 ppb	Growing season average

Foliar fluoride concentration of 30 ug/g ppm fluoride in forage

Ozone: photochemical oxidant (ozone)

0.10 ppm	Hourly average, not to be exceeded more than once a year;

Nitrogen dioxide:

0.05 ppm	Annual average
0.17 ppm	Hourly average, not to be exceeded more than once a year;

Carbon Monoxide:

9 ppm	8-hour average, not to be exceeded more than once a year;
17 ppm	Hourly average, not to be exceeded more than once a year;

Lead:

1.5 ug/m^3	calendar quarter average

Hydrogen Sulfide:

100 ppb	Hourly average, not to be exceeded more than once a year

In considering the effects of the various types of air pollution found in Montana, it must be kept in mind that it is virtually never possible to say with certainty that a given pollutant concentration causes a particular effect on health or the environment. The most that can be said is that the effect is "associated" with such conditions.

The effects known to be associated with pollutants found in Montana are as follows:

Sulfur dioxide at concentrations known to occur in Montana has been demonstrated elsewhere to contribute to increased death rates, irritation of the throat and lung, and injury of throat and lung tissues. Lesser amounts of sulfur dioxide are required to cause such effects when particulate matter also is present. Agricultural crops are threatened by concentrations higher than those necessary to threaten human health. It appears that there is no level of sulfur dioxide low enough to assure that no health or environmental effects will occur.

Mixtures of sulfur dioxide and particulate matter in the air have been found to adversely affect victims of bronchitis and asthma. Particulate material consisting primarily of larger particles travels only a relatively short distance from the source and is primarily a nuisance. It has little or no known effect on health or the environment.

Photochemical pollutants at elevated levels are related to eye irritation and headaches. Healthy adults and people with asthma are adversely affected at short term photochemical concentrations known to be found in Montana. Exercise during exposure apparently decreases the amount of photochemical pollutant required to bring on the various health effects. Various types of plants found in Montana show damage at photochemical oxidant exposures present in the state. Like most other pollutants, photochemical exidants seem to be more potent when they are mixed with other pollutants.

Carbon monoxide is absorbed into the body from concentrations in the air. In the body, it is associated with a steep decline in the ability of the blood to circulate oxygen. Traffic concentrations in some places in Montana cities leads to corbon monoxide concentrations sufficient to threaten sensitive persons, such as pregnant women, children, and those with an impaired heart, lungs, or blood vessels.

Nitrogen dioxide also is generated in combustion processes, such as in industrial facilities and automobile engins, and in photochemical reactions. Nitrogen dioxide is both a participant and a result in the photochemical generation process.

Research indicates that the effects of nitrogen dioxide are most pronounced in children and persons with asthma or chronic lung disease. Damage to plants has been observed at nitrogen dioxide levels known to occur in Montana.

Contamination of the soil by lead pollution results in the threat of lead poisoning to children in Montana. Testing of children in East Helena, near the ASARCO lead smelter, found that seven percent of the children had concentrations of blood lead high enough to be associated with potentially serious effects. Studies elsewhere show that the blood lead levels found in the East Helena children can be related to decreased performance on intelligence tests. The elevated blood lead was believed to be related to the amount of lead deposited from the air in the soil surrounding the smelter. The standard was developed by relating various concentrations of blood lead in children to ambient lead levels.

Hydrogen fluoride is the most toxic of all Montana pollutants to plants. It apparently causes no significant effects on humans until concentrations far above those necessary to severely damage plants and threaten foraging animals. Several studies have shown hydrogen fluoride to be associated with heavy damage to plants in Montana. Approximately 69,000 acres of Glacier National Park have experienced elevated fluoride levels resulting from the Anaconda aluminum plant emissions. Ambient fluoride concentrations sufficient to threaten plants and animals are found both around the Anaconda plant and the Stauffer Chemical phosphate plant at Ramsay.

The Anaconda Aluminum plant is expected to meet the proposed standards with equimpment now scheduled for installation.

For hydrogen sulfide, the only major source, the Hoerner-Waldorf pulp mill, appears to be in compliance with the proposed standard.

Several of the other pollutants for which standards are required result primarily from the use of automobiles and trucks. These include nitrogen dioxide, carbon monoxide, and photochemical oxidants. Nitrogen dioxide and carbon monoxide are released directly from such sources. Photochemical oxidants are created in the air by the interaction of sunlight and various "precursor" pollutants generated by automobiles and trucks.

There is a variety of methods for controlling these pollutants. The catalytic converters in newer cars, for example, remove most carbon monoxide from exhaust gases. The technology for controlling nitrogen dioxide and other photochemical precursors coming from engines is less well developed, and the control methods usually recommended involve traffic control plans to disperse automobiles over a wide area to prevent concentrations of the pollutants. Less operation of automobiles and smaller engines are two other approaches.

A substantial amount of the particulate matter released into the air in Montana comes from area sources, such as dusty roads, timberland slash burns, and farmers' fields. The best strategy for controlling such sources is not always clear. Dusty roads can be controlled by sprinkler trucks, and open burning can be restricted to periods of favorable weather, but other sources are more difficult.

Other types of sources causing concern are open mines, such as the Berkely pit in Butte and the coal strip mines of eastern Montana. There is a variety of "housekeeping" measures available to reduce emissions from these sources.

Air pollution in Montana comes from a wide variety of sources, everything from dusty roads and trash burning barrels to major industrial facilities. The bulk of the potentially hazardous pollution comes from major sources, with the capacity to emit at least 100 tons of pollution per year. There are approximately 50 of these in Montana, but this impact statement is concerned primarily with the fourteen among these most likely to be affected by new ambient standard. The fourteen are: The Anaconda Aluminum plant at Columbia Falls, the Hoerner-Waldorf pulp and paper mill in Missoula, the Anaconda Copper smelter at Anaconda, the Stauffer Chemical Company phosphate plant at Ramsay, the Berkely Pit copper mine in Butte, the ASARCO lead smelter in Helena, the Cenex, Conoco, and Exxon refineries in the Billings--Laurel area, the Montana Power Company's J. E. Corette power plant in Billings, the Montana Sulphur and Chemical Plant in Billings, the Colstrip and Sidney coal fired generators, and coal strip mines.

It is estimated that adopting the proposed standards would save Montana approximately $17.1 million a year. A major portion of this saving, approximately $14,400,000, would result from medical costs from reduced sulfur dioxide concentrations. Reduction of plant damage form sulfur dioxide and fluoride would contribute another $2.2 million a year. Decreased materials damage would save approximately $500,000.

By comparison, the total capital and operating costs for polluting industries to meet the standards would be approximately $4.6 million per year, straight line depreciation.

I. INTRODUCTION

In the fall of 1977 the Montana Department of Health and Environmental Sciences (DHES) was considering enforcement action against some Montana industries for violations of the administrative regulation on Ambient Air Quality Standards (ARM 16-2.14(1)-S14140). This rule had been on the books more than ten years and had been regarded during that time as an enforceable regulation. During the research in preparation for the enforcement action, however, it was discovered that there was some doubt whether the Board of Health and Environmental Sciences ever intended for the standards to be directly enforceable. In adopting the ambient air quality standards, the board said:

> The board, in adopting these standards, intends them to be goals and guidelines and so interprets the legislative intent of the word "standards" in section 69-3909 of the Clean Air Act of Montana.

Further clouding the intent of the board was the discussion by board members during the debate on the standards, when they seemed to favor enforceability. A phrase in the same rule referred to the standards as "maximum permissible concentrations." This and other language in the rule led some to believe the standards were intended to be legally enforceable (Brown, 1977).

When this confusion was brought to the attention of the board in December, 1977, the board members said they wanted Montana to have enforceable ambient air quality standards and directed the Air Quality Bureau to propose an administrative rule containing new, enforceable standards.

It had been ten years since the ambient standards were adopted, so it was decided that scientific information developed in the interim should be evaluated and new standards proposed. This impact statement contains the findings of that review and tells how the information reviewed was used to develop proposed standards in line with the best current knowledge.

The board, in proposing to adopt ambient air quality standards, is well within the philosophical and legal framework prevailing in Montana. The 1972 Montana Constitution provides in Article IX:

> (1) The state and each person shall maintain and improve a clean and healthful environment in Montana for present and future generations.
> (2) The legislature shall provide for the administration and enforcement of this duty.
> (3) The legislature shall provide adequate remedies for the protection of the environmental life support system from degradation and provide adequate remedies to prevent unreasonable depletion and degradation of natural resources.

The Clean Air Act of Montana (R.C.M. 1947, 69-3901, et seq.) provides specific legal authorization for enactment of ambient air standards. Section 69-3909(5) of the act requires the Board of Health and Environmental Sciences to "establish ambient air standards for the state." Policy objectives which must be considered by the board in adopting these standards are stated in Section 69-3905(1) of the act:

> It is hereby declared to be the public policy of this state and the purpose of this Act to achieve and maintain such levels of air quality as will protect human health and safety, and to the greatest degree practicable, prevent injury to plant and animal life and property, foster the comfort and convenience of the people, promote the economic and social development of this state, and facilitate the enjoyment of the natural attractions of this state.

Similarly, "air pollution" was defined in the act to mean:

> The presence in the outdoor atmosphere of one or more air contaminants, in a quantity and for a duration which is or tends to be injurious to human health or welfare, animal or plant life, or

property, or would unreasonably interfere with the enjoyment of life, property, or the conduct of business.

In considering the adoption of an air quality standard, five questions need to be answered. First, is it feasible to accurately measure the ambient concentrations or emissions of the pollutant? Second, is there enough information available on the effects of the pollutant to legally support a standard? Third, is the pollutant present in sufficient concentrations to merit regulation? Fourth, should the standard be enforceable or merely a guideline? Finally, how stringent should the standard be? Inherent in this last question are several other questions. For example, what should the standard protect and what margin of safety should be set to compensate for pollution effects not currently recognized?

One obvious source of help in answering these questions is the experience of the federal government under the federal Clean Air Act, which required adoption of federal ambient air quality standards.

Federal Ambient Air Quality Standards

In 1970, the Congress amended the federal Clean Air Act to require the Environmental Protection Agency to adopt national ambient air quality standards. The amendments specified that these were to be the minimum acceptable standards; the states clearly retained the authority to adopt more stringent standards. The federal act required two separate standards for each pollutant, a "primary" standard designed to protect human health, and a "secondary" standard to protect property, the environment, and other elements of the "public welfare." The current federal standards are listed in Table I-I. Ambient air quality standards adopted by several representative states and foreign countries are listed in Appendix B.

The federal act required each state to develop a plan (State Implementation Plan) to reduce pollution in the state enough to meet the standards.

TABLE I-I NATIONAL AMBIENT AIR QUALITY STANDARDS

	Primary Standard	Secondary Standard
Particulate		
24-hour average	260 ug/m^3 *	150 ug/m^3
Annual geometric mean	75 ug/m^3	60 ug/m^3***
Sulfur dioxide		
3-hour average		0.5 ppm**
24-hour average	0.14 ppm	
Annual average	0.03 ppm	
Carbon Monoxide		
1-hour average	35 ppm	35 ppm
8-hour average	9 ppm	9 ppm
Ozone		
1-hour average	0.08 ppm	0.08 ppm
Nitrogen dioxide		
Annual average	0.05 ppm	0.05 ppm
Lead		
Calendar quarter	1.5 ug/m^3	1.5 ug/m^3

---

* Micrograms per cubic meter (ug/m^3) is a measure of the mass of a pollutant in one cubic meter of air. A microgram is one-millionth of a gram or about 1/28,000,000 of an ounce. A meter is 39.37 inches or 100 centimeters.

** Parts per million (ppm) is a measure of the volume concentration of one gas mixed with another gas. For example, one ppm should mean one gallon of a pollutant is diluted in one million gallons of air.

***This value is not a standard but is a guideline to be used in assessing state implementation plans.

The federal act requires the primary standards to be met within three years after EPA approves that state plan. The secondary standards are to be met in a "reasonable time." The definition of a "reasonable time" depends on how expensive and complex it is to control the pollutants, but is not to be more than a few years after the primary standards are met.

If a state does not submit a plan to achieve national standards, the EPA will impose its own. If the state does not submit a plan or if it fails to enforce the plan, various federal enforcement mechanisms are available to the EPA. For example, if the state did not submit an enforceable carbon monoxide control plan, federal highway funds could be withheld.

The federal act does not authorize the issuance of a compliance order or the collection of a penalty for violations of ambient standards. Instead, federal ambient standards are enforceable only against the state, which is required by law to implement the federal act. If a state refuses to act, EPA will adopt a rule of its own prescribing the maximum amount of pollution a source can release to the atmosphere, in order to reduce pollution concentrations to meet the federal ambient standards. In other words, in the federal law, ambient standards are a guide to the selection of a proper emission regulation.

In Montana, the DHES has maintained that ambient air quality standards should be enforceable directly against a source. Although the DHES always has embraced emission regulations as the primary method of achieving compliance with ambient standards, it also has insisted that the Montana Clean Air Act provides other enforcement procedures, specifically, the issuance of an administrative enforcement order against a source for violations of ambient standards, and/or the initiation of a civil or criminal action in district court for the collection of a penalty when a source violates an ambient standard.

Prevention of Significant Deterioration

Amendments to the Clean Air Act in 1977 established two new rules which have almost the same effect as national ambient air quality standards. The Prevention of Significant Deterioration (PSD) regulations have a significant effect on the allowable level of sulfur dioxide and particulates in many areas of Montana. Under the PSD regulations, areas of Montana may be designated as Class I, II or III according to the amount of air quality deterioration the local residents will accept. Class I is defined as an area where almost any change of air quality would be considered significant, and therefore not permitted. In a Class II area, the increased air pollution normally accompanying moderate, well-controlled growth would not be considered significant. In a Class III area, air pollution can increase by levels equal to one-half the federal standards before it is considered significant. Of the three classes, Class III allows the greatest deterioration of air quality. Pollution increases allowed under PSD regulations must not exceed the most stringent federal standard. Table I-II, shows the present federal standards and the increase permitted in each classification.

Currently, most of the state is classified as Class II. The National Parks, most wilderness areas and the Northern Cheyenne Indian Reservation are all classified as Class I. The Fort Peck Indian Reservation and the Flathead Indian Reservation have announced their intention to ask for Class I designation. By following strict federal procedural rules, the state may change the classification of the present Class II areas to either Class I or Class III. No changes are now pending review.

TABLE I-II PREVENTION OF SIGNIFICANT DETERIORATION
ALLOWED INCREASE ABOVE 1974 POLLUTION LEVELS

	Federal Standard	Class I	Class II	Class III
Particulates				
24-hour(ug/m^3)	150	10	37	75
Annual	75	5	19	37
Sulfur Dioxide				
3-hour (ppm)	0.5	0.01	0.20	0.27
24-hour (ppm)	0.14	0.002	0.035	0.07
Annual (ppm)	0.03	0.0008	0.008	0.016

In many areas of Montana the sulfur dioxide levels are virtually zero. That means most of the state is under the sulfur dioxide limits of either Class I or Class II. These levels are substantially more strict than the federal ambient air quality standards. In most rural areas of Montana the annual average particulate concentrations have been about 15 to 20 ug/m^3, with the 24-hour levels about 100 ug/m^3. For such areas with a Class II designation, the allowed PSD increments would establish a permitted particulate level of approximately 40 ug/m^3 for the annual and approximately 140 ug/m^3 for the 24-hour average. Additional pollutants are expected to be placed under the PSD regulation in the future, thereby setting maximum permitted increases for them also.

Visibility Protection

The 1977 Amendments also provided for protection of visibility in Class I areas. The new provisions require that the states adopt implementation plans that include emission regulations for any source that "may reasonably be anticipated to cause or contribute to any impairment of visibility" in a Class I area. New power plants, manufacturing facilities, and other sources of pollution cannot be built unless they can meet the emission regulations. Essentially, Congress has said no visibility deterioration will be permitted in these areas.

Considerations in Setting Montana Ambient Air Quality Standards

The federal law requires that the primary standard must "protect the public health" with an "adequate margin of safety." The secondary standards must protect "the public welfare" from "any known or anticipated adverse effects." The public welfare is defined to include "effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

Montana law similarly requires standards be adopted which will "protect human health and safety," and to the greatest degree practicable, protect property and the environment.

Numerous questions can be raised on the meaning of these statutory requirements. For example, what is "the public health?" Does it just include obvious illness, or does it include very subtle changes in body functions? Should only the middle-aged person in good health be protected or also the very young, the very old, or the already ill person? Similarly, should only agricultural crops be protected or should the analysis consider long-term effects on the ecosystem? These questions were considered in developing the standards recommended in this document.

Who should be protected?

Who should be protected from air pollution? When air quality standards were first proposed by Governor Goodwin Knight of California in 1954, he urged that they should include the effects on "elderly chronic invalids" (Knight, 1955). The California standards adopted in 1959 were intended to respond to the needs of "persons in the population who are most sensitive to air pollution effects" (Calif. Dept. of Public Health, 1959). In writing the 1970 amendments to the federal Clean Air Act, Congress took the same tack. The Senate committee emphasized that "included among those persons whose health should be protected by the ambient standard are particularly sensitive citizens such as bronchial asthmatics and emphysematics" (U.S. Senate, 1970). This indicates that aggravation of pre-existing diseases is to be considered. It is important to note that the aggregate of these "sensitive groups" may constitute a sizeable fraction, perhaps even a majority, of the population. Although the federal perspective is not binding on Montana, the precedent for "who should be protected" is substantial and was adopted by the Montana Ambient Air Quality Standards Study.

Human Health Effects

What constitutes a "health effect?" One frequently used classification divides health effects into the clinical (directly observed changes in body function), behavorial (changes in the central nervous system or mental ability), subjective ("I know what I don't like and I don't like it"), and body burden (increased concentration of a pollutant in the body with no immediately apparent effect).

Many clinical effects occur at commonly occuring ambient air pollution levels. Eyes can be irritated, throats become scratchy, or asthma aggravated. Sometimes there may be immediate, visible tissue damage or even death. These effects are generally due to short-term exposures to relatively high pollutant concentrations and are termed "acute" effects. Effects brought on by long-term exposures to very low levels of pollution are called "chronic" effects. They may be as significant as cancer or as subtle as anemia. A short-term exposure also may cause damage which shows up much later as a chronic affect.

The body is quite capable of making measurable changes in its chemistry without either significantly reducing the body's stores of energy or producing any side effects. While some changes in body chemistry or body burden may be important warning signals of a serious health threat, other changes may be a useful adaption to a stress, at no long-term cost to the body. To properly evaluate the importance of such subtle effects it is necessary to know the eventual results they are associated with.

Medical research on the effects of pollution on humans is difficult, and it is hard to be certain a given effect is a threat to health. Therefore, persons setting pollution standards usually consider all such effects at the risk of erring on the side of caution. In the east European countries and the Soviet Union ambient air quality standards usually are based on measurements of altered body functions such as modifications in brain waves or secretions of various body chemicals (enzymes). In the United States, at least one federal air quality standard (carbon monoxide) was based on a similar subtle change in central nervous system activity.

Experiments With Air Pollution and Human Health

Three types of experiments are used to define the impacts of air pollutants on human health: animal studies, clinical studies, and epidemiological studies.

Animal studies are valuable for determining the effects of pollutants on laboratory animals under controlled conditions in experiments that would be too hazardous with human subjects. Animal experiments allow the use of high pollutant concentrations and examination of affected tissues. They make possible the repetition of experiments and the determination of relationships between given pollution levels and the effects observed. Although the findings of these studies are not directly applicable to humans, there is a general understanding that responses found in experimental animals most likely will cause similar effects in humans.

Clinical health studies are used for more direct determination of air pollution responses in humans. The advantage of this method is that precise levels of pollutant can be administered under consistent study conditions. Because the experiments usually use volunteers, often college students, it is difficult to experiment with long-term or repeated exposures. There is little doubt these studies understate the effects on the general population.

The epidemiological studies evaluate health responses under ambient conditions common to the human environment. Testing for low level effects in humans often is possible only through statistical survey in cities alike except for their pollution levels. These studies relate pollution levels to illness and death rates. The Senate report quoted earlier categorizes such effects as an element of the "public Health" that is to be protected.

It said:

> "ambient air quality is sufficient to protect the health of persons whenever there is an absence of adverse effect on the health of a statistically related sample of persons in sensitive groups from exposure to the ambient air." (U.S. Senate, 1970)

Epidemiological studies are especially useful in identifying a sensitive group or detecting an unusual type of illness or cause of death that might be associated with pollution.

Environmental Effects

Air pollution may also injure plants, modify the climate, reduce visibility, destroy stone monuments, or contribute to other changes in the environment. The most significant environmental damage is often a reduction in crop yields. This is potentially very important in Montana, with agriculture the largest primary industry and production of wood products the fourth largest. Together these two pollution-sensitive industries account for almost 55 percent of the total primary employment in the state. Other environmental damage from air pollution can adversely affect tourism, the second largest primary industry in Montana.

All pollutants released into the atmosphere eventually return to the earth. For some pollutants the cycle lasts only a few days, while for others it may take ten days to several weeks to complete. Most of the pollution will be taken up by plants, through either their leaves or roots. The remainder will build up in the soil or run off into rivers and lakes. Some pollutants fall directly into the water. Both fish and aquatic plants can be affected by air pollution. Wildlife can be affected by eating polluted forage which contain pollutants or by a loss of forage from air pollution damage to plants.

Air pollution can result in the death of some plant tissues without killing the whole plant. This can reduce the growth rate (especially significant for timber) or the amount of crop yield. As in the study of human health effects, it is desirable to be able to detect the onset of such effects in plants before such important adverse events occur. Changes in the ability of the plant to absorb and convert food and sunlight are often monitored as indicators of air pollution effects.

Laboratory experiments on plants are usually conducted under ideal circumstances, with the plant receiving just the right amount of water, light, and fertilizer. This type of experiment may be valid for certain agricultural crops which are tended with almost the same care. By contrast, plants growing under natural conditions are often on the edge of survival even without the added insult of pollution. Thus laboratory experiments may significantly understate the affects that pollution will have on uncultivated plants.

The Threshhold Concept

With most pollutants an increase in concentration produces a parallel increase in adverse effects. Low concentrations produce small effects, larger concentrations produce larger effects. As concentrations of a given pollutant increase, an increasing percentage of the population will show the effects produced by that pollutant. By contrast, some pollutants have what is termed an "effect threshhold," which is defined as a level below which there is no effect on human health or the environment. Below the threshhold, any number of persons or any portion of the environment may be exposed for any period of time, even a lifetime, with no adverse consequences. An ambient standard maintaining the level of a given pollutant below the threshhold thus would prevent damage by that pollutant.

For some pollutants, however, there may be no threshhold. In such a case, even the smallest measurable amount of the pollutant may cause an adverse effect. For these substances the tolerable exposure is zero. Most substances which cause cancer are believed to be "no-threshhold" compounds, although a certain time of exposure may be necessary to cause the effects (Yanyshera and Automonov, 1976).

Margin of Safety

The federal law requires consideration of a "margin of safety" in setting the primary standard. This is a precaution to protect the public from the possibility that the effects of a given pollutant may be more serious than they currently are known to be.

The margin of safety used in setting a standard reflects the risk society is willing to accept. The margin used in setting the federal ambient air quality standards ranged approximately from one to three for various pollutants (Finklea, 1973). A factor of three in this case indicates a standard was set at one-third the level known to cause adverse effects. By comparison, engineers use factors of approximately 10 in designing simple systems in aircraft. The Food and Drug Administration claims to use a factor of 100 in regulating use and availability of hazardous materials in food and medicine. The Nuclear Regulatory Commission uses factors ranging from 100,000 to 1,000,000 when calculating the safety of nuclear facilities. Determining what safety factor should be used is a matter of policy which must reflect the willingness of society to accept risk. In considering what ambient standards should be set for Montana, the staff of the Air Quality Bureau has followed the federal precedent by using a factor ranging over a continuum from approximately one to approximately three. That is to say that for some pollutants, the standard proposed will be set at the lowest level where effects are known to occur, while for other pollutants the proposed standard will be set at a level only one-third the level known to cause effects. The margin used for any given pollutant depends primarily on the seriousness of the danger from that pollutant and on the difficulty of controlling it.

It is a matter of dispute whether there must be absolute certainty about the effects of a pollutant before a standard can be set. One court case interpreted the federal Clean Air Act to require only that the pollutant "may reasonably be anticipated to endanger public health or welfare." The court said. "regulatory action may be taken before the threatened harm occurs; indeed the very existence of such precautionary legislation would seem to demand that regulatory action precede and, optimally, prevent, the perceived threat" (Ethyl Corp. vs E.P.A., 1976). The definition of "air pollution" in the Montana Clean Air Act contains similar language.

Economic Considerations

The Montana Clean Air Act requires that the formulation of an air quality standard designed to protect the environment may include consideration of what pollution controls are economically "practicable" to install. Impacts on energy supply and consumption, non-air pollution environmental effects, and other social factors also may be considered. The act stipulates that health must be protected, but grants the board considerable discretion in setting standards intended to protect more than human health (Brown, 1978).

A benefit-cost analysis for each standard is not required. Such analyses are extremely difficult and sometimes impossible to do. On the cost side, the actual installation costs and the probable operating and maintenance costs can be calculated for a specific control procedure on a specific pollutant at a specific plant, but extending the estimate to include further controls, or other pollutants or controls on a different plant usually requires seemingly endless analysis.

Furthermore, it can be exceptionally difficult to estimate the value of benefits that would result from various pollution controls. It is hard to measure the amount of illness or environmental damage caused by a particular pollution concentration and it is almost impossible to get agreement on the dollar value of health or life. The value of health is more than the cost of medical services or lost wages. There is no accurate estimate of what a person would pay to avoid a given illness, much less to avoid premature death.

High costs may be imposed on the environment even where there is no large human population to be directly affected. Many pollutants travel far from their sources, causing significant effects at considerable distances. The costs of some potential impacts are large but not easily determined. For example, it is difficult to calculate what losses would result if air pollution reached concentrations sufficient to reduce sunlight and reduce the length of the growing season.

Even if it were possible to measure total benefits and total costs for each possible standard, the problem of balancing control costs with the benefits of reduced pollution would not be solved. If further controls were installed to the point where costs and benefits of the increased control were equal, further controls still would be required to completely eliminate the potential for damage to health and property.

The benefits of not spending the additional money to further reduce pollution fall to some extent on society as a whole, but the major benefits go to the employees and stockholders in the company, and to the buyers of the product and the sellers of raw materials. The burden, by contrast, is borne by those exposed to the pollution. Seldom do the burdens and benefits fall on the same people. Thus the inclination to reduce emissions is demonstrably stronger in the persons who carry the burden without the benefit than in those who reap the benefits without the burdens.

Analyzing the long=term economic impacts of environmental degradation is more difficult than evaluating the economic effects of ill-health. It may take damaged forests or farms several hundred years to regain full potential after severe pollution by airborne contaminants. Benefits occurring so far in the future have little economic value to present generations. The current generation has a clear responsibility as a trustee of the environment for future generations. Where the land has been damaged, fairness to future generations may require that the current generation bear the burdens of alleviating the damage and preventing further degradation.

Striving for the least cost to society as a whole does not result in social fairness to all members of the society. The Anglo-American tradition, by contrast, emphasizes the importance of the individual above the importance of society and stresses social fairness above economic rewards. The Montana Legislature acted in this tradition when it required protection of the public health and safety from air pollution regardless of the cost.

Monitoring Air Pollution

Ambient air quality standards are designed to protect property, the environment, and the public health. The questions often asked are, what property, what part of the environment, and what aspect of the public health is to be protected?

The standards are not intended to protect the health of indoor workers on the job, so it would not be proper to measure and enforce the standards inside an industrial facility. It is clear that measurements should be taken in residential and commercial areas where many people may be exposed during their normal daily activities. It is not clear if the standards should be measured and enforced in areas that the public seldom visits or on other property owned by the owner of the source. Even though no one would be likely to spend extended periods in a farmer's field, it would be entirely proper for pollution to be measured there. The lone farmer has the same right to protection of his land and family as the resident of the busy city.

It is sometimes suggested that air quality standards not be enforced on land owned by the owner of the pollution source. It is argued that such enforcement would result in the owners of the source bearing more expense than necessary to alleviate pollution damage since it often would be cheaper to buy the land affected by pollution than to control the emissions. This argument is complicated by two difficult problems. First, a system of purchasing polluted land might lead eventually to a purchase-lease back scheme where the polluting industry could own a nearby "company town." This could have many undesired social consequences. Second, such a system would not meet the state's obligation to protect wildlife, which belongs to the state and not to an individual property owner. If substantial areas of rural land were owned by the owner of a pollution source and closed to monitoring by control authorities, the state's interest in wildlife could not be protected.

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II. AIR POLLUTION IN MONTANA

Section A of this chapter provides general information about air pollution in Montana. Section B discusses the 13 largest industrial sources of air pollution in Montana.

II.A. STATEWIDE PERSPECTIVES

Air pollution sources fall into two categories, point sources and area sources. Point sources are relatively well defined sources which tend to emit their pollutants from a fairly small area, such as a smokestack or industrial complex. Most point sources are industrial plants.

There are approximately 50 major point sources of air pollution in Montana. Thirteen of these have been selected for discussion here. These thirteen contribute more than 85 percent of Montana's industrial sulfur dioxide emissions, more than 85 percent of the industrial particulate emissions, and virtually all the emissions of fluoride. These are the sources most likely to be affected if the state adopts enforceable ambient air quality standards. These plants, their emissions, and the extent of their pollution control programs are described in Section II-B. The sulfur dioxide and particulate emissions from major point sources are listed in Table I-V in Appendix A.

Increasingly, pllution in Montana is caused by people going about their day-to-day activities rather than industrial plants. All else being equal, increased population in an area leads to increased pollution. A principal cause is increased automobile use, and increases in automobile-generated pollution, including road dust. Cities and rural areas with growing population thus may anticipate increased pollution. Table VI in Appendix A shows projected population increases in counties with significant sources of air pollution. County figures were used rather than town figures in order to include the increasingly urbanized portions of formerly rural areas in the counties.

Table II.A.-I summarizes the measurements of ambient air quality in Montana cities. This table lists the highest levels recorded in 1977 for each pollutant during various averaging times. More detailed information can be found in Appendix A.

Area and Mobile Sources

Agriculture

The disturbance of soil by cultivation sometimes leads to problems with windblown dust. Another source of air pollution from agricultural operations is the burning of stubble fields and ditch banks. The Air Quality Bureau minimizes smoke concentrations from agricultural and other open burning by issuing burning permits only when weather conditions are favorable for good dispersal of the smoke. Agricultural operations emit an estimated 20,000 tons of particulate each year (Gelhaus *et al*., 1978).

Dusty Roads

Road surface materials in unpaved roadways are ground into dust by traffic, and are likely to become airborne by wind. Unpaved roads

TABLE II.A-I SUMMARY OF SELECTED AMBIENT AIR QUALITY DATA (1977)

Pollutant and Averaging Time	Missoula	Anaconda	East Helena	Great Falls	Billings	Colstrip
Sulfur Dioxide						
Max. 1-hr. (ppm)	0.05*	2.00	0.88	---	0.20	0.10
Max. 24-hr. (ppm)	0.02*	0.78	0.26	---	0.02	0.05
Annual (ppm)	0.00*	0.038	0.019	---	0.002	0.00
Particulates						
Max. 24-hr (ug/m^3)	676*	161	192	869	165	95
Annual Geo. Mean	82*	44	103	52	58	28
Settled Particulate (gm/m^2)	---	---	---	10.7	---	---
Visibility						
Annual (Miles)	**	---	---	65	60	---
Carbon Monoxide						
Max. 1-hr. (ppm)	31.6	---	---	23.1	19.2	0.0***
Max. 8-hr. (ppm)	25.9	---	---	14.4	13.1	0.0***
Ozone						
Max. 1-hr. (ppm)	0.075*	**	---	---	0.08	0.050***
Nitrogen Dioxide						
Max. 1-hr. (ppm)	0.098*	**	---	---	0.180	0.130
Annual Arith. Mean	**	**	---	---	0.029	0.029
Hydrogen Sulfide						
Max. 30 min. (ppm)	0.0561	---	---	---	---	---
Annual Arith. Mean	0.0006	---	---	---	---	---
Total Hydrocarbons						
Max. 1-hr (ppm)	7.92	---	---	---	26.40	3.50***

* = 1978 Data
** = Insufficient Data
--- = No data
***= 1975 to 1976 Data

Source: Gelhaus et al., 1978; Trijonis and Shapland, 1978; Unpublished Air Quality Bureau data, 1978.

contribute more than 696,700 tons per year of airborne particulate. This is about 90 percent of all Montana particulate emissions (Gelhaus et al., 1978). The majority of the particles are relatively large and heavy and tend not to travel far. The large particles are mainly a nuisance and have little effect on health. Paving is the only permanent cure for dusty unpaved roads.

Paved roads may sometimes become dirty and contribute to airborne dust problems. Such conditions are best controlled by cleaning roads periodically. Several cities are purchasing street cleaning equipment in order to reduce suspended particulate levels.

Open Burning

The burning of logging slash is a major source of particulates and other cumbustion generated pollutants in Montana. The burning of railroad right of way is another source of combustion pollutants, as is the burning of leaves and other waste in residential areas. All these forms of burning are controlled under a cooperative smoke management program in which state and federal agencies and independent industries participate. The program allows burning under favorable weather conditions. The emissions from open burning are estimated to be approximately 24,500 tons/year (Gelhaus et al., 1978).

Coal Strip Mines

Coal strip mines are a significant source of particulate emissions in eastern Montana.

Five major companies operate strip mines in Montana. There are many indications that others will be opened in the near future. It is difficult to precisely determine emission levels from any given mine, or to generalize the emission characteristics for mines of this type. Each mine has its own characteristic emission sources which are not necessarily duplicated in any other mine.

In some mines draglines are used to strip the overburden, while others use trucks and shovels. Some revegetate and apply water and mulch immediately after spoils are leveled and others do not. Some use outdoor coal storage and others use shelters. Some have long haul roads and others short.

One example, not necessarily typical of such mines, is the Western Energy Mine at Colstrip and the nearby Peabody mine. Table II.A-II shows estimated 1974 emissions and estimated 1980 uncontrolled emissions of the Peabody mine and the various areas of the Western Energy mine.

TABLE II.A-II COLSTRIP AREA COAL MINE PARTICULATE EMISSIONS FOR 1974 AND PREDICTIONS FOR 1980 IF NO CONTROLS ARE USED (Tons/yr)

	WESTERN ENERGY					PEABODY ACTIVITY			
	Area A		Area B	Area E	Pit 6			TOTAL	
	1974	1980	1980	1980	1974	1974	1980	1974	1980
Coal Removal	20	122	122	180	60	50	100	130	452
Overburden Removal	142	839	839	741	412	294	588	848	3007
Scraper Activity	35	104	104	104	104	35	70	174	382
Haul Roads	45	271	271	298	133	83	166	261	1006
Vehicle Exhaust	31	69	69	69	69	31	69	131	276
Storage	22	132	132	116	65	neg.	neg.	87	380
Wind Erosion	54	540	540	540	355	194	292	603	1912
Blasting	73	73	73	73	73	73	73	219	292
Area Total	422	2140	2140	2039	1261	860	1348	2543	7667

Source: PEDCo 1977.

A recent report specifies several methods that can be used to reduce emissions from these mines. These methods could be applied with similar results to other mines with similar sources. The methods are shown in Table II.A-III with the estimated efficiency and consequent emission reductions.

TABLE II.A-III SUGGESTED EMISSION CONTROLS FOR COLSTRIP AREA COAL MINES

Control Method	Estimated Efficiency	Estimated Emission Reduction (tons per year)
Cover coal stockpiles	100	380
Soil stabilization in active mining areas	80	1530
Haul road speed control and dust suppression	50	503

Source: PEDCO, 1977.

Other estimates provided by Montana Power Company estimated the efficiency of haul road watering to range from 34 to 43 percent efficient. The revegetation of disturbed areas was estimated to be 80 percent efficient in reducing dust emissions.

Montana Power data from Colstrip shows an annual mean particulate level of 107 ug/m^3 and a highest 24-hour average of 428 (second highest reading-379 ug/m^3). The monitoring station operated by the Air Quality Bureau in the area does not show high particulate levels because it is on top of a hill several hundred feet high and is not exposed to emissions from mining activity. This monitoring site was chosen to reflect power plant emissions, rather than low level mine emissions.

Home Heating

Home heating can be a significant source of air pollution. Fireplaces and wood stoves emit particulates and oil-fired furnaces are a source of sulfur dioxide, originating from the sulfur in the fuel. The Air Quality Bureau does not permit the burning of fuel oil with more than one pound of sulfur per million BTU fired.

Cars and Trucks

Cars and trucks are the major mobile pollution sources in Montana. The most important pollutant emitted from cars and trucks is carbon monoxide. Total estimated carbon monoxide emissions from mobile and industrial sources in Montana are shown in Table VII in Appendix A. Although trasportation related carbon monoxide accounts for a relatively large amount of the total amount emitted, and the resulting ambient concentrations are significant because they occur along streets where humans may be affected. Ambient carbon monoxide concentrations in Montana cities are shown in Table VIII in Appendix A. A significant reduction of carbon monoxide emissions from mobile sources could be achieved by reducing the number of sources, or the amount they are driven. The catalytic converters in new cars eliminate nearly all carbon monoxide from exhaust gases. Better tuning of engines would result in some reduction, and this might be achieved by state inspection of auto emission control equipment.

Another important pollutant from cars is lead, from the burning of leaded gasoline. Lead emissions vary from 40 to 100 micrograms of lead per mile traveled (EPA, 1977). This amount is decreasing and will continue to decrease as a greater percentage of cars on the road use nonleaded gasoline. There is no reliable estimate of lead emissions from automobiles in Montana.

Hydrocarbons and nitrogen oxides are two other important pollutants from mobile sources. These two pollutants can react in the presence of sunlight to form ozone and other photochemical pollutants. The estimated emissions of nitrogen oxides and hydrocarbons from area and industrial sources are shown in Table VII in Appendix A. Ambient levels of nitrogen dioxide measured in Montana are listed in Table IX and concentrations of ozone in Table X in Appendix A.

The primary control of hydrocarbons from motor vehicles is by engine tuning and recovery of vapors from the gasoline tank and carburetor. Effective controls for nitrogen oxides from gasoline engines are not generally available and depend on successful development of the "three-way" catalyst. Reductions in the emissions of nitrogen dioxides from diesel engines are even more problematic. To obtain significant reductions in nitrogen oxide emissions from motor vehicles it may be necessary to control the number of diesel-powered vehicles or to reduce the amount they are driven.

Photochemical Pollutant Generation

Certain pollutants are generated by chemical reactions among other pollutants in the air. These transformations are called "photochemical reactions," because they result from sunlight reacting with airborne pollutant mixtures. Pollutants participating in the reactions are called "precursors," with the main ones being hydrocarbons* and nitrogen oxides.

These compounds originate primarily from cars, trucks, oil refineries and other fuel burning equipment. They are most concentrated in urban areas. The toxic products of photochemical reactions consist mainly of ozone,nitrogen di and lesser amounts of other compounds collectively referred to as oxidants. Nitrogen oxides are both precursors and products of the process.

The mechanism by which photochemical smog is produced is extremely complicated and not completely understood. Researchers have found that certain minimum levels of sunlight and the various precursors are necessary to initiate the process.

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* The term hydrocarbon is meant to include all hydrocarbon organics capable of participating in the atmospheric oxidant formation process.

Figure II-1 is a representation of the chemical changes that occur when a mixture of hydrocarbons and nitrogen oxides is exposed to sunlight in sufficient proportions to result in the production of photochemical oxidants. The curves of Figure II-1 indicate the overall photochemical process, which consists of two distinct reaction stages occurring consecutively. During the first stage, nitric oxide is converted into nitrogen dioxide without any appreciable buildup of ozone or other oxidants. The second stage starts when almost all of the nitric oxide has been converted into nitrogen dioxide and is characterized by rapid accumulation of ozone and other oxidant and nonoxidant products.

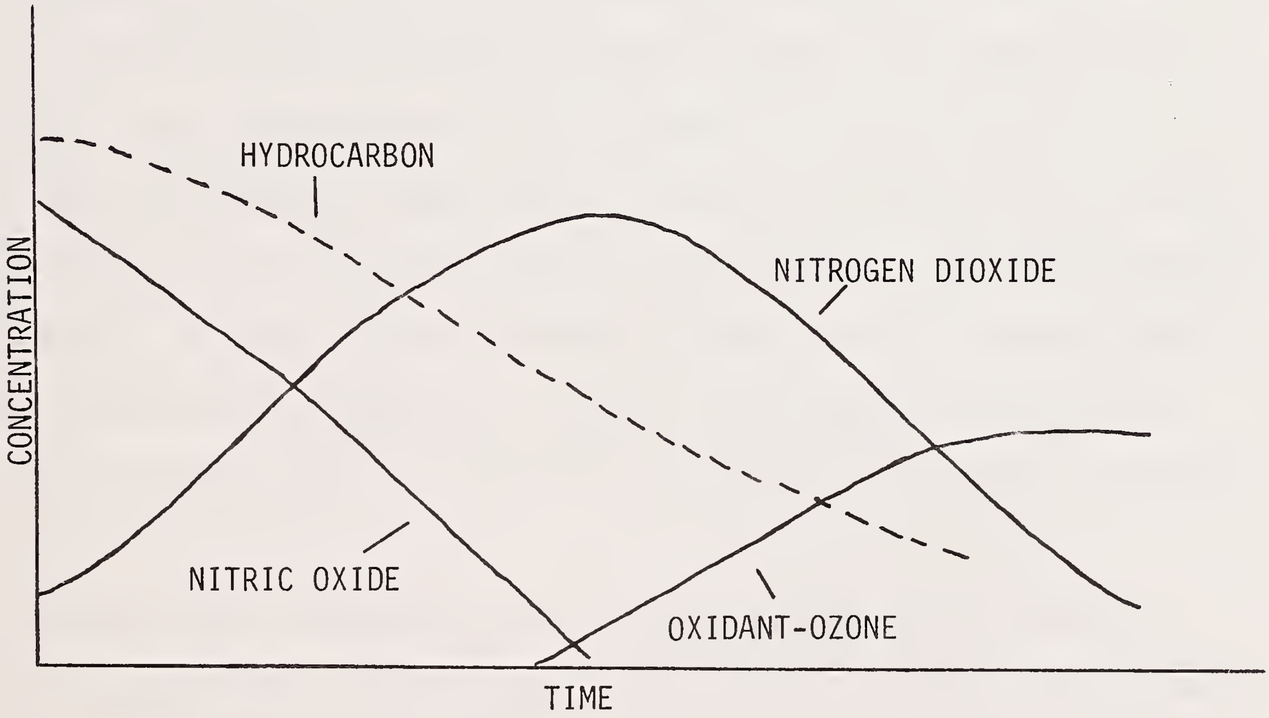

Figure II-1 Chemical changes occuring during exposure of hydrocarbon-nitrogen oxide-air systems to sunlight

Three photochemical pollutants are discussed in this impact statement: ozone, PAN, and nitrogen oxides. Substantial amounts of nitrogen oxides also are generated by industrial and transportation sources. Hydrocarbons are discussed because of their importance as photochemical precursors.

The total amounts of photochemical pollutants generated in Montana is not known. Ambient levels of ozone are shown in Table X in Appendix A.

Literature Cited

Environmental Protection Agency. 1977. Air Quality Criteria for Lead. EPA-600/8-77-017.

Gelhaus, J. W., J. Schneider, and M. D. Roach. 1978. Annual Air Quality Data Summary for Montana. Montana Air Quality Bureau. 89 pp.

PEDCo, 1977. Southeastern Montana Coal Resource AQMA Analysis: prepared for U. S. EPA, Region VIII.

II.B ENVIRONMENTAL CONDITIONS AROUND MAJOR POLLUTION SOURCES IN MONTANA

This section describes thirteen industrial sources of air pollution in Montana, their control programs, and the condition of the ambient air and the environment around each. A summary of the emissions from these plants is contained in Table II.B-I.

The Missoula Vicinity

Missoula is at the north end of the Bitterroot Valley about five miles east of the confluence of the Bitterroot and Clark Fork Rivers, at an elevation of approximately 3000 feet. The Clark Fork Valley extends about 20 miles west-northwestward from Missoula. The Bitterroot Valley runs southward about 70 miles. The town is surrounded by mountain ranges which markedly affect its climate and pollution levels.

The prevailing air flow aloft over western Montana is from the west to southwest during spring and summer and from the west and northwest during the winter. Moist air from the Pacific loses much of its moisture as it passes over the Bitterroot range west of Missoula, so the town gets an annual average of only about 12 to 15 inches of precipitation. The climate therefore is semiarid. The most rain comes during May and June, with average rainfall of about two inches in each month. There is considerable farming in the area. Farm products grown in the valley are listed in Table XI in Appendix A.

In winter, the continental divide, approximately 70 miles east of Missoula, protects the town from much of the severely cold air which moves down the continent from the arctic. This shielding effect deflects many cold waves from the Missoula area.

TABLE II.B-I ESTIMATED EMISSIONS FROM POINT SOURCES OF AIR POLLUTION AND PRESENT DEGREE OF CONTROL

	SULFUR DIOXIDE		PARTICULATE		LEAD		FLUORIDE		NITROGEN OXIDES		HYDROCARBONS	
SOURCE	t/yr	%	t/yr	%	t/yr	%	t/yr	%	t/yr	%	t/yr	%
Anaconda Aluminum	2,200	0	1440	77			455	90				
Anaconda Copper	281,750	34	4780	95	179	95						
Berkeley Pit	207	unk	4023	33								
ASARCO Lead	14,000	76	418	96	48	95						
CENEX Refinery	10,380	unk	398	unk					540	unk	unk	unk
Conoco Refinery	3,198	unk	263	unk					1194	unk	unk	unk
Exxon Refinery	9,800	unk	735	unk					1401	unk	unk	unk
Hoerner-Waldorf Paper	365	unk	760	98					1008	unk	115	unk
Montana-Dakota Utilities, Sidney	2,372	15	430	98								
Mont. Power Co. (Colstrip 1 & 2)	5,326	75	618	99					7000	unk		
Mont. Power Co. (Corette)	9,986	10	1124	97					6757	unk	94	unk
Montana Sulphur	1,530	97							4	unk		
Stauffer Chemical	208	unk	99	96			62	50				

unk=unknown or unmeasurable

Source: Gelhaus, et al., 1978; , Constant et al., 1977;

The surrounding mountains profoundly affect the air pollution levels found in the Missoula area. Protected from wind and weather systems, the valley experiences frequent temperature inversions of sufficient strength and duration to cause elevated concentrations of trapped pollutants.

Data collected by the Air Quality Bureau in 1971 and 1972 showed that the average inversion height between October 1, 1971 and January 31, 1972, was 1500 feet above the valley floor. More relevant to ambient pollution levels is the mixing height, which is the elevation to which air movement causes pollution dilution. Data collected at the Missoula airport show an annual average mixing height of 939 feet. At times however, the mixing height dropped as low as 350 feet. There are extended periods of calm.

Pollution sources in Missoula include numerous industrial and area sources, as listed in Table V in Appendix A, and a growing population with its associated area and mobile sources.

Figure 1 in Appendix A shows the Missoula area with major sources and ambient monitors labeled. Ambient measurements of hydrogen sulfide from the Missoula area are listed in Table XII in Appendix A. The only source likely to be affected by the adoption of ambient standards is the Hoerner-Waldorf pulp and paper mill.

The Hoerner-Waldorf Pulp and Paper Mill

The most recent figures available show that in 1976 this mill used 1,535,000 wet tons of sawdust and wood chips in producing 404,296 tons of kraft paper.

The current estimated pollution emissions from the Hoerner-Waldorf mill are shown in Table II.B-I. Details of the plant operation and process and the pollution control history of this plant is available in the environmental impact statement prepared in response to proposed expansion of the plant (AQB, 1974A).

Pollution control devices installed since 1974 are listed in Table XIII of Appendix A. No further controls are planned.

The Columbia Falls Vicinity

Columbia Falls is at the northeast corner of the Flathead Valley at 3037 feet elevation. Glacier National Park lies east and north of the town, with the nearest point about nine miles northeast. The mountains surrounding the valley range in elevation from approximately 5000 feet to 7000 feet. The topographical features contribute to weather conditions conducive to pollution problems in the area. The high mountains to the east block the severe cold waves from Canada, and help give the area a milder climate than the territory across the continental divide, 40 miles to the east. The snow stays well into early summer in the mountains, cooling moist air coming from the west and stimulating rainfall.

Data from the Glacier Park International Airport a few miles south of Columbia Falls show that the prevailing wind from March through September is from the southeast. This wind blows off Flathead Lake and is caused by the land surfaces warming faster than the surface of the lake. This wind sometimes is quite gusty, and often reaches 20 mph or more. Nighttime winds flow from east to west out of Badrock Canyon but currents are not strong. Emissions from the aluminum plant are not diluted by the wind as much at night as during the day, so nighttime emissions accumulate in the air near the plant. The pollution rises at night but is not buoyant enough to go over the mountains. During the day, prevailing winds from the south carry accumulated nighttime emissions over the saddle in Teakettle Mountain toward

Glacier Park. This effect is most pronounced around mid-morning; later in the day the accumulation of nighttime pollutants has been removed and only that day's emissions move over the mountain.

Annual precipitation at the airport averages 15.42 inches, of which a substantial portion is contained in the annual average of 67 inches of snowfall. The precipitation is sufficient to encourage considerable farming. Crop species grown in the county are listed in Table XI in Appendix A. The agricultural character of the land is changing as the growing population of the area converts agricultural land to residential subdivisions. The highly desirable surroundings in this area contribute to the high rate of population growth, one of the highest in the state.

Wood products are a major source of employment in the area, with several sawmills located in the Columbia Falls vicinity. The wood products industry produces air pollution from a number of sources, including the open burning of logging slash, and disposal of waste wood in tepee burners.

The aluminum plant is the only major source likely to be affected by ambient standards. Location of the aluminum plant and monitoring stations is shown in Figure 2 in Appendix A.

The Anaconda Aluminum Plant

This plant uses electrolytic reduction process in which alumina ore from Jamaica is dissolved in a molten bath of aluminum and fluoride-sodium salts in a carbon-lined steel crucible. Suspended above the crucible is a large carbon block which acts as an electrode to deliver a high intensity electric current through the bath into the crucible. The current reduces the alumina ore to molten aluminum, carbon monoxide and carbon dioxide. At plant capacity, 392,040 tons of alumina are processed into 180,000 tons of aluminum per year.

Pollutants produced in this process include both gaseous and particulate fluorides, other particulates, and sulfur dioxide. Emissions from the plant are shown in Table II.B-1.

When the plant was built in 1955, it relied on mechanical devices called multiclones for particulate removal. A type of wet scrubber was used to remove gaseous emissions. In 1968 it was estimated that 7500 pounds of gaseous and particulate fluorides were released into the air from the plant each day. By 1972 the old scrubbers were replaced with more efficient venturi type scrubbers, and fluoride emissions were reduced to approximately 2500 pounds per day, where they remain.

A 99.7 percent efficient dry scrubber now scheduled for installation is expected to reduce fluoride emissions by about 475 pounds per day. A scheduled switch to a different type of paste in the anode is expected to reduce nonfluoride particulate emissions by approximately 50 percent. Planned modifications in the process will improve control of the gases produced and lower the fluoride concentration in the bath, thus reducing fluoride and other emissions. This reduction is expected to bring emissions down to less than 850 pounds per day.

Fluoride emissions could be reduced to 400 pounds per day by the installation of ten scrubbers to clean gases escaping through the roof of the buildings. There is no present plan to install these additional scrubbers. Considerations affecting the need for further controls are discussed in Section II.C.

Visible plant injury caused by fluoride emissions from the aluminum facility has been observed over 69,000 acres, much of which was in Glacier National Park (Carlson and Dewey, 1971). Elevated ambient fluoride concentrations have been found as far away as the mountain northeast of Lake

McDonald in Glacier Park, a popular tourist spot in the park. Carlson and Dewey reported that a total of 71,670 acres of Glacier Park had elevated ambient fluoride levels.

Although fluoride emissions from the plant have been reduced substantially since the Carlson and Dewey study, monitors continue to show elevated fluoride levels at some stations, as shown in Table XIV in Appendix A. Some monitoring stations in Glacier Park show annual average ambient fluoride concentrations increasing over the period since their installation in 1975 (Hall, 1978). Measurements of the concentration of fluorides in vegetation near the Anaconda Aluminum Company plant are reported in Table XV in Appendix A.

The Anaconda-Ramsay-Butte Vicinity

The topography of this area is highly conducive to air pollution problems with industrial point and area sources located in mountain valleys where emissions frequently are trapped by temperature inversions or other stable air conditions. The elevation of populated areas ranges from approximately 5200 feet at Anaconda and Ramsay to approximately 5600 feet at Butte. Anaconda is located at the south end of the Deer Lodge Valley, with Ramsay 15 miles east on Silver Bow Creek and Butte another nine miles east in the Silver Bow drainage.

Near Anaconda inversion conditions prevail approximately 40 percent of the time on an annual average. About 45 to 50 percent of the inversions occur in the winter, with another 30 percent in the spring. Inversion frequencies at Anaconda are among the highest in the nation. There is no comprehensive weather information available from Butte or Ramsay.

There are three significant sources of air pollution in this area. The Anaconda Company's copper smelter at Anaconda is the source of approximately 80 percent of all the sulfur dioxide emissions in the state, including emissions from both industrial point sources and area sources. The Berkeley open pit mine is a source of particulates. The Stauffer Chemical Company's elemental phosphorous plant at Ramsay is a fluoride source.

Agriculture in this area is hindered by high elevation and the effects of air pollution. Near Ramsay plants and animals are affected by fluoride emissions from the phosphate plant. In the Anaconda vicinity, air pollution from the smelter has led to heavy metal concentrations in the soil, making it difficult for plants to grow (Taskey, 1972). Heavy metal concentrations measured in soil near Anaconda are shown in Table XVI in Appendix A. Crops grown in the Anaconda-Ramsay-Butte area are listed in Table XI in Appendix A.

There are few trees at the lower elevations except along stream and river banks where there are willows, aspen, and cottonwoods. At higher elevations there are thick coniferous forests of Douglas fir, engelman spruce, lodgepole pine, rocky mountain juniper and common juniper.

This region differs from most other industrial areas in the state in having a declining population. This decline is not likely to have much effect on air pollution in the area, however, because the net population loss is small, and any decline in pollution would be more than overshadowed by fluctuations in the industrial operations.

Figures 3 and 4 in Appendix A show the Butte and Anaconda area with major sources and monitoring sites labeled. Table XVII in Appendix A shows the ambient sulfur dioxide levels for the area in 1977. Table XVIII in Appendix A shows the ambient concentration of suspended particulate for the area in 1977. Table XX shows ambient air fluoride levels around the phosphorous plant at Ramsay and fluoride levels in vegetation in the same area.

The Anaconda Copper Smelter

Copper ore, concentrates and precipitates are brought to the smelter for processing from Nevada, British Columbia, and Butte. The smelter has a rated capacity of 35 million pounds of copper per month. Yearly sulfur dioxide and particulate emissions are shown in Table II.B-1. Nearly all the sulfur dioxide generated in the process comes from the converters and furnace. The plant generates a total of about 1170 tons per day of sulfur dioxide. About 34 percent of this, or 398 tons, is controlled with a remaining emission of 772 tons. Most of the captured sulfur dioxide, about 383 tons per day, is removed by a new sulfuric acid plant which produces approximately 565 tons per day of sulfuric acid.

There are many sources of particulates in the smelting process. The particulates generated contain many heavy metals, including zinc, lead, arsenic, copper and cadmium. Many of these escape uncontrolled, but over 99 percent of those drawn into the flue system are removed by the new baghouse.

Total particulate removal efficiency for the plant is estimated to be 95 percent. The most current estimate of particulate emissions is 4780 tons per year, an average of 14 tons per day. Current emissions are high enough to cause violations of various ambient regulations. Ambient concentrations of lead measured near the Anaconda plant are shown in Table XIX.

Major pollution controls installed in recent years include the new baghouse and acid plant. The acid plant has a production capacity of 990 tons per day of sulfuric acid. Sulfur dioxide emissions could be further controlled by installation of additional acid plant capacity. Control efficiency of the sulfur dioxide control equipment now installed or planned for installation is estimated at 86 percent.

Berkeley Pit

The Berkeley Pit mine in Butte is a sort of hybrid point source and area source, with approximately nine square miles of potential dust emission area. The haul roads in the pit are the major source, with lesser emissions resulting from power shovel operations, road maintenance, blasting, fuel combustion in trucks, and mineral waste dumpting. Haul road emissions are reduced considerably by wetting down with eight 85 ton water trucks. The use of more water trucks would further reduce particulate emissions, but the purchase of these is at the discretion of the Anaconda Company, which may use any of a number of methods to meet emission standards. The tuning of the many diesel engines operating in the pit to minimize their individual emissions is another possible method of reducing the total emissions.

Stauffer Chemical Company's Elemental Phosphorous Plant

This plant extracts elemental phosphorous from phosphate rock. Pollution is emitted from numerous sources within the plant. The most significant pollutants released are gaseous and particulate fluorides. Approximately 70 to 80 percent of the fluorides are gaseous. Numerous control devices have been installed to reduce emissions, with others scheduled for installation. Data from Stauffer show that 96 percent of total particulates generated are currently captured, with an additional two percent expected to be captured with devices scheduled for installation.

Current control of total fluorides is approximately 50 percent. Measures scheduled for installation are expected to increase this to 75 percent. There appears to be a technological possibility of increasing total control to 85 to 95 percent. Current estimated emissions are shown in Table II.B-1.

The Helena-East Helena Vicinity

Helena and East Helena are located about four miles apart at approximately 4000 feet elevation at the south end of the Helena valley. The valley is approximately 25 miles wide and 35 miles long. The average elevation of the surrounding mountains is approximately 6700 feet.

Winds in the area are predominantly westerly about half of the time. Calm conditions prevail about nine percent of the time. The geography of the Helena Valley is ideal for the formation of strong and persistent temperature inversions. Shelter from the surrounding mountains contributes to low wind speeds and calm conditions. Cold air draining into the valley from the mountains under calm conditions contributes to the formation of pollution trapping inversions. Inversions are present approximately 30 percent of the time. Temperature soundings taken with aircraft during the cold months show the occurrence of persistent inversions up to 1800 feet deep.

Agriculture in the valley is being replaced rapidly by subdivision development. Crops grown in the county are listed in Table XI in Appendix A. The largest industrial source in the valley is the ASARCO lead smelter in East Helena.

Figure 5 in Appendix A shows the location of the ASARCO plant and ambient pollution monitors.

ASARCO Lead Smelter

The ASARCO lead facility in East Helena is a custom smelter accommodating both foreign and domestic ore. In 1977 it processed approximately 209,137 tons of ore from Canada, Australia, South America, Montana and other states. Production for that year was 54,455 tons of lead bullion which was sent to a secondary smelter in Omaha for further refining.

Pollutant emissions from the smelter include carbon monoxide, sulfur dioxide and particulates. Approximately 30 tons per day of particulates are drawn into ducts and flues, and 98 percent of these are captured by pollution control devices. Additional particulates (less than one ton a day) escape from process equipment, blow off ore and material piles, or otherwise escape capture. These escaping particles have a substantial content of lead, arsenic, and cadmium. The smelter emits an estimated 300 pounds per day of lead and 36 pounds per day of arsenic. Ambient air concentrations of lead are reported in Table XIX in Appendix A.

The total amount of sulfur dioxide generated by the plant is approximately 153 tons per day. Approximately 75 percent of this is removed by pollution control devices now in place. Up to 90 percent control is possible through redesign of process equipment and installation of further controls. Ambient sulfur dioxide concentrations around East Helena are shown in Table XVII in Appendix A. Eighty-three tons per day of carbon monoxide are released to the air without control.

Pollution control devices now in place include a new 500 ton per day acid plant for converting sulfur dioxide into sulfuric acid, a 99 percent efficient baghouse for removing particulates from the low strength sulfur dioxide streams, and an electrostatic precipitator for removing particulates from high strength sulfur dioxide streams. Three baghouses installed in 1903 remove an estimated 96 percent of the particulates from blast furnace off-gases. Each of these has a relatively low wooden smokestack.

These three stacks are scheduled to be replaced by a 375 foot stack, which is expected to reduce ground level sulfur dioxide and carbon monoxide concentrations.

The Billings-Laurel Vicinity

Billings and Laurel are located about 16 miles apart on the Yellowstone River in south central Montana. By Montana standards the two towns have pollution problems of major proportions. Several industrial sources, a rapidly growing population and topography favoring the buildup of pollution all contribute to the pollution potential.

The population of Yellowstone County grew an estimated 14 percent between 1970 and 1976, and this growth rate is expected to continue. The Yellowstone county population for 1970 and projected 1980 population are shown in Table VI in Appendix A.

This area is on the border between the great plains and the Rocky Mountains and has a climate with some of the characteristics of each of the two regions. The National Weather Service classifies the climate as semi-arid, but irrigation and a favorable annual distribution of the precipitation makes it possible to raise a variety of crops. Crops grown in Yellowstone County are listed in Table XI in Appendix A. Average annual precipitation is slightly more than 14 inches. The Yellowstone Valley in this area is oriented generally southwest to northeast, and the prevailing winds are from the southwest.

Geography is a major contribution to the pollution potential. Laurel is on generally flat ground and is more or less exposed to whatever surface winds there are, which helps reduce pollution problems.

Billings, by contrast, is situated between sandstone cliffs that shelter it from surface winds to some extent, contributing to the relatively high pollution levels sometimes found there. The elevation of Billings ranges from 3100 feet to 3600 feet. Industrial pollution sources in the vicinity include a refinery in Laurel; and in Billings, two refineries, a coal-fired power plant, and a sulfur plant. The location of these sources and the ambient pollution monitors is shown in Figures 6 and 7 in Appendix A. Ambient sulfur dioxide particulate levels for the vicinity are shown in Tables XVII and XVIII in Appendix A.

Farmers Union Central Exchange (Cenex) Refinery in Laurel

In 1977, this refinery processed 12,679,367 barrels of crude oil, producing propane, gasoline, No. 1 heating fuel, road oil, No. 2 diesel fuel, butane and asphalt. Sixteen tons of sulfur was produced daily. Approximate pollution emissions are shown in Table II B.-1.

Pollution controls in place include a carbon monoxide boiler on the fluid catalytic cracking unit (FCC) and a Claus elemental sulfur plant to clean the emissions. The Claus sulfur plants produce elemental sulfur from the sulfur impurities originating in the feedstock crude oil. The FCC and steam boilers are major sulfur dioxide emission points Both could be controlled by scrubbing. New FCC catalysts for sulfur dioxide control are under development and may be available soon. Particulates released in the process could be controlled with readily available particulate control equipment. Cenex has agreed to modify several items of process equipment to reduce sulfur emissions from the refinery. The company also will extend the 100 foot stack currently serving the sulfur plant to 199 feet, and modify it to serve both the existing sulfur plant. A new 199 foot stack will replace twin 50 foot stacks venting the crude preheat system. Cenex also is considering raising six other stacks ranging from 90 feet to 125 feet tall to a uniform 199 feet, but no final decision has been made on this.

The Exxon Refinery in Billings

In 1977 this plant processed 13,967,003 barrels of crude oil into the usual petroleum products. Pollution emissions are shown in Table II.B-I.

Pollution controls in place include various devices which remove sulfur from the gas used to fuel the refining process. Further emission control could be achieved by scrubbing the coker and FCC emissions.

Conoco Refinery in Billings

In 1977 this refinery processed 16,775,633 barrels of crude oil into the usual petroleum products. Table II.B-I shows approximate emissions.

Pollution controls in place include various devices which remove sulfur from the gas used to fuel the refining process.

Montana Sulphur & Chemical Company

This company uses gases containing hydrogen sulfide which are piped from the Exxon and Conoco refineries to produce a variety of sulfur products. Total hydrogen sulfide gas from Exxon is approximately 42 tons per day. Total hydrogen sulfide from Conoco is approximately 33 tons per day.

These gases are used to make approximately 73 tons per day of flake sulfur, powdered sulfur, raw sulfur and purified liquid hydrogen sulfide. Pollution emissions from the plant are shown in Table II.B-I. The pollution control equipment in place is an incinerator in the stack.

The Montana Power Company's J. E. Corette Coal-Fired Power Plant

In 1977, the plant burned 625,697 tons of coal from the Western Energy strip mine at Colstrip. Also burned was 358,938,000 cubic feet of natural gas, for flame stabilization in the boiler. The plant has a rated capacity of 180 megawatts (Mw) but the usual maximum load is 165 Mw. The average load for 1977 was estimated at 144 Mw. Net electrical production for 1977 was 1,097,255 Mw hours.

The sulfur in the coal used during the year was estimated at 5236 tons, or 0.84 percent. Potential sulfur emissions are reduced by the removal of high sulfur pyrite impurities before the coal is burned. Approximately five to ten percent of the sulfur in the coal is removed through the pyrite rejection.

Particulate emission control is an electrostatic precipitator with an efficiency of 97.5 percent. Current estimated emissions are shown in Table II.B-1.

The Colstrip Vicinity

The town of Colstrip lies on the northern great plains, east of the Rocky Mountains. The elevations in the vicinity of Colstrip range from less than 2900 feet along Rosebud Creek about 12 miles east of Colstrip to over 4400 feet 20 miles to the south. The elevation of Colstrip itself is about 3200 feet. The topography is developed on the essentially flat lying sediments of the Fort Union formation which contain most of the coal in the region. The rolling upland is cut by streams, producing narrow valleys, buttes, and some fairly level land. Most of the level areas are well-developed flood plains of streams.

Colstrip lies within the upper Missouri drainage basin, on the East Fork of Armells Creek, which drains directly into the Yellowstone River. The Rosebud Creek drainage runs from southwest of Colstrip to the east and then north into the Yellowstone River. The Yellowstone River flows from west to east.

The population density of the Colstrip vicinity always has been quite low. In 1970 the population of Rosebud county was 6032 persons, mostly ranchers, scattered over 5042 square miles. The projected 1980 population is 10,100. The increase reflects the influx of workers associated with coal development in the area. The population of Colstrip has risen from approximately 200 to approximately 2000 since the approval of generating units 1 and 2 in 1973.

Ranching and other agriculture comprise the bulk of the land use in the Colstrip area. The ranches are generally a combination of grazing and dry-land crop farming. Soil and moisture are such as to permit some tillaged and irrigated agriculture in the valley bottoms, with crops such as alfalfa hay, wheat, oats, and barley.

There are some forested areas in the vicinity and a portion of the Custer National Forest lies southeast of Colstrip. Some logging is done in this area, but the scale is small compared to western Montana. Coal is mined in two places near Colstrip--the Western Energy mine just outside town and the Peabody coal mine a few miles south.

Total precipitation averages about 15 inches per year. A year long weather study at Colstrip indicated there were 215 inversions during the period. These inversions lasted from 10 hours to almost three days. The short inversions normally develop at night when air nearest the ground cools below the temperature of air higher in the atmosphere. These nighttime inversions usually are weak and are broken when the sun warms the ground which in turn warms the lower layers of air. Inversions of this type thus do not normally last beyond the warmest period of the day.

The short inversions were most common in August, September, and October.

The 14 inversions lasting more than 24 hours occurred during the winter months. There were seven in December, three in January, four in February. The longest inversion lasted 67 hours, and occurred in December. Several other inversions lasted more than two days.

The data seem to indicate the inversions were strongest during the fall and early winter with over 24 percent of December having extremely stable atmospheric conditions which would tend to increase ambient pollution levels.

The only major point source in the vicinity is the two Colstrip 360 Mw coal-fired generators. Emissions from these are shown in Table II.B-1. Figure 8 in Appendix A shows the eastern Montana pollution monitors and the Colstrip generating site.

The Montana Power Company's Colstrip Power Plant

This plant consists of two 360 Mw plants which together burn up to 3,000,000 tons of coal per year to generate electricity. Both particulates and gases are controlled with venturi scrubbers. Alkaline fly ash produced in the boilers is combined with water for use as a scrubbing medium. Particulate removal efficiency is 99.5 percent, and the alkaline scrubbing medium provides sulfur dioxide removal of approximately 75 percent. No further control devices are planned for this facility.

The Sidney Vicinity

The town of Sidney is located approximately two miles north of the Yellowstone River about six miles west of the North Dakota border in Richland County. The elevation is approximately 1900 feet. This is primarily flat farming country, with an overall climate much like that of Miles City, Scobey and other areas of eastern Montana.

Prevailing winds are from the west with rigorous weather systems out of Canada. Ambient air data from Sidney is scarce, and pollution problems apparently are few.

The area is sparsely populated, with a 1970 population of 9837 spread over 2103 square miles. There is considerable farming in the county, both dry land and irrigated. Total agricultural production in the county is shown in Table XI in Appendix A.

The only significant industrial point source of air pollution in the vicinity is the 50 Mw Montana-Dakota Utilities Company coal-fired generator near the Yellowstone River, approximately two miles south of Sidney.

The Montana Dakota Utilities Company's Coal-fired Power Plant

In 1977 this plant burned 312,551 tons of coal with an average 0.47 sulfur. A total of 11,664,000 cubic feet of natural gas were burned for flame stabilization.

Total sulfur dioxide generated was approximately 2791 tons, of which approximately 15 percent was removed in the venturi wet scrubber, resulting in a total sulfur dioxide emission of 2372 tons per year. The scrubber is primarily for control of particulates, with an efficiency of approximately 98 percent. If further emission controls were required, an estimated 70 percent efficiency could be achieved for sulfur dioxide control with the use of a lime or limestone scrubber. The scrubber currently in use could not be adapted for such use. Operating changes in the present scrubber would improve particulate collection to an estimated 98.6 percent but with a considerable increase in energy cost.

Literature Cited

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III. ENVIRONMENTAL IMPACTS OF AIR POLLUTANTS

This section discusses the effects on human health, agricultural and other plants, livestock and other animals, ecosystems, and materials of the eight pollutants for which air quality standards are being proposed.

III. A. SULFUR OXIDES AND PARTICULATES

Introduction

Sulfur dioxide is a colorless gas which irritates membrane tissues in the nose, throat and lungs. In either water or air it can be changed to sulfuric acid or sulfate. The highest ambient levels of sulfur dioxide in Montana have been measured near the Anaconda copper smelter. This smelter is the source of more than 80 percent of sulfur dioxide emissions in Montana. Very little data is available on sulfate levels in Montana Additional information on the sources and ambient measurements of sulfur dioxide and sulfates in Montana can be found in Section II.B and Appendix A.

Particulates include both liquid droplets and dry particles with a wide variation in size and origin. The highest ambient concentrations of particulates in Montana appear to be related primarily to fugitive dust from various services. Additional information on the sources and ambient measurements of particulates in Montana can be found in Section II.B and Appendix A.

Sulfur dioxide and particulates at the concentrations occasionally recorded in Montana have been observed to have adverse effects on human health. Sulfur oxides irritate the throat and lung and contribute to increased respiratory disease. The health effects of sulfur dioxide are more pronounced in the presence of a moderate amount of fine particulate. The persons most sensitive to sulfur oxides and particulate exposures are those with asthma, children and elderly bronchitis patients.

Adverse effects on vegetation, materials and the environment also are known to occur at levels of sulfur oxides and particulates measured in Montana. The growth, size and yield of some plants are reduced. Insects and micro-organisms are affected. Metals and building materials are corroded and soiled. Increases in rainfall acidity due to sulfur dioxide have substantial impacts on soil and forest ecosystems. In Scandinavia, acid rains have caused the loss of whole biological communities including plants, small animals and fish from lakes and river systems.

Sulfur dioxide and particulates are discussed together for two reasons. First, sulfur dioxide converts in the atmosphere to sulfate, which is believed to be one of the most significant airborne particulates. Second, fine particulates can carry sulfur dioxide deep into the human lung, increasing the potential for adverse effects on health.

Sulfur dioxide can dissolve in a water droplet and form sulfurous acid (Hocking and Hocking, 1977). If a reactive chemical, such as a metal or part of an organic molecule also is dissolved in the droplet, a "sulfite" may be formed. Sulfur dioxide also can adhere to the surface of a particle Ozone and other oxidants, and even sunlight can convert the sulfur dioxide in a droplet, on a particle, or in the air to sulfur trioxide or sulfuric acid. The kind of acid or sulfate formed from sulfur dioxide in the atmosphere is determined by atmospheric conditions such as humidity and air contaminants. Several theories have attempted to describe the chemical processes, and it has been suggested that various particulate pollutants may participate (as catalysts) in the reaction (Larson and Harrison, 1977; Frieberg, 1974). The rate of conversion from sulfur dioxide to sulfate has been measured under varying conditions from one percent per hour to over 16 percent.

The effects of particulates on human health depends on their size and composition. The mechanisms working to alter the composition of particulates in polluted air are so many and varied that each individual particle in a particulate mass may have a different composition. Among the mechanisms is the condensation and collection of gaseous pollutants and small particles on still smaller particles. This collecting of other particles and gases may lead to a high concentration of metals, organic substances and other chemicals on particle surfaces.

The mechanisms do not necessarily distribute pollutants evenly in a mass of particles, and health hazards may be increased if a relatively small number of particules contain the bulk of the contaminants. This is especially true for radioactive pollutants.

The compounds most commonly found in the fine particles are sulfur dioxide, lead and bromine from automobile gasoline additives, nitrates from nitrogen oxides emissions, and carbon and organics from evaporated organic vapors and combustion. The common constituents of soil, such as calcium, iron, aluminum and silicon, are found mostly in coarse particles. These may have originated from the processing of rock and minerals or from soil dust raised by wind or automobiles. Fragments of plants, pollen, and other natural organic materials will be found among the coarse particles. Tiny fragments of automobile tires can make up a significant fraction of the coarse particles in urban areas.

The fine and coarse particles also differ in their physical behavior. The coarse particles generally will fall out of the air very close to their source. As a result, they are most frequently found in urban areas or near a source, such as an unpaved road. Fine particles are more nearly the size of atmospheric gas molecules, so they remain suspended much longer, sometimes up to a week. Given this much time, the wind can blow particles halfway across the continent. In this way fine particles are spread over a much larger area than coarse particles. Rural areas may have a higher percentage of fine particulate than urban areas, even though the total amounts of particulate are less.

The tendency of fine particles to move with the air also causes fine and coarse particles to behave differently when inhaled. Large particles will collect on surfaces in the nose and throat while smaller particles will flow into the windpipe (tracheal-bronchial tree), and the lung. The nose is a very effective coarse particle filter. Very large particles are captured in the outer parts of the nostril. Smaller particles are caught in the deeper areas of the nose and throat. If a person is at rest and breathing through the nose, almost all particles larger than 10 micrometers will be deposited in the nose and throat. However, light exercise and breathing through the mouth will cause as much as 35 percent of the 10 micrometer particles to pass through the throat into the trachea. Only four to five percent of this amount will enter the lung (Task Group on Lung Dynamics, 1966). Particles caught in the rear portion of the nose are moved to the throat and swallowed. If they are soluble they will pass directly into the bloodstream.

Fine particles are more likely than coarse particles to flow around the obstructions in the airways and reach the lungs. They also are more likely to be exhaled. Half the particles inhaled will be exhaled. About 20 percent of the one micrometer particles are filtered out in the nose and throat, while five percent are retained in the tracheal-bronchial tree and 25 percent in the lung. Almost all the 0.1 micrometer particles will enter the lung and about 50 percent of them will remain there* (Task Group on Lung Dynamics, 1966). Once deposited in the deep lung, particles may remain for long periods. This long retention time in the lung makes it possible for insoluble compounds in or on fine particles to be transferred to the blood to a much greater extent than if they are swallowed into the stomach. Thus, about ten times more insoluble substance, such as lead, enters the bloodstream from particles in the lung than from the same mass of particles in the stomach (Natusch and Wallace, 1974).

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* The percentages quoted are calculated for conditions at sea leavel. In high altitude areas such as Butte, the lung deposition may be 20 percent greater (EPA, 1978).

Several methods have been used to collect and measure suspended particulate. A system developed in England draws a small stream of air through a long tube and a filter. The amount of particulate in the air is measured by shining a light on the dirty filter paper and reading the amount of light reflected. The degree of reflection depends mainly on how dark the filter becomes from the particulate adhering to it. Both the amount and color of the dust influence the amount of light reflected. The light reflected is compared to the reflection from a specified amount of a laboratory-generated "standard" smoke in order to read the result as a mass concentration (in micrograms per cubic meter - ug/m^3). Obviously, if the measured dust is not identical to the standard smoke, the apparent mass concentration may have no relation to the actual mass concentration in the air. This measurement method is referred to as British Smoke (BS). A similar method was used in Los Angeles for a short time. In that instance the reflectance was reported in KM (coefficient of mass) units.

A modification of the British Smoke system by the American Iron and Steel Institute (AISI) measures the amount of dust by shining a light through the filter paper. The amount of light transmitted depends on the amount and color of dust deposited. A standard reduction of light transmitted is defined as one CoH (Coefficient of Haze) unit. It has been calculated that one CoH represents a concentration of 1450 ug/m^3 of white limestone dust, but only 310 ug/m^3 of grey fly ash or 65 ug/m^3 of black coal dust. Two CoH represents 104 ug/m^3 of coal dust (adapted from Droege and Clark, 1974).

The standard American particulate sampling device is the high volume (hi-vol) sampler, which draws a large volume of air through a paper filter which is then weighed. This method measures total suspended particulate (TSP). The hi-vol will capture more large diameter particles than the British Smoke or AISI devices. Thus it will record a greater mass concentration in most situations than will the other two instruments.

It is difficult, if not impossible, to compare measurements from the BS, AISI, and hi-vol methods. Attempts have been made to compare them and some conversion factors have been developed. In one study a British smoke filter and a hi-vol were operated side by side in London from 1955 to 1963 (Commins and Waller, 1967). Data reported in that study have been analyzed by one of the authors and the equation

$$\text{TSP } ug/m^3 = 119 + 0.824\ (\text{BS } ug/m^3) \quad (r=.72)$$

is reported to be valid up to about 700 ug/m^3. Above that level the readings were found to be equivalent. A direct conversion for the old Los Angeles system used by the California Air Resources Board is TSP (ug/m^3) = (38+2) (KM + 1.1).

Several groups have attempted similar studies for the AISI device. Although a conversion of one CoH = 125 TSP ug/m^3 is frequently quoted, there is no evidence of a consistent relationship from place-to-place or from season-to-season in the same place. For example, an extended study at seven sites in Washington State found the conversion factor to vary from 136 to 508 ug/m^3/CoH depending on the color of the dust (Droege and Clark, 1974).

The data summarized in this section is stated as it appears in the original reports. No attempt has been made to convert British Smoke or CoH measurements into equivalent TSP values.

Human Health Effects

Sulfur oxides and particulates together form a pollutant complex more hazardous to human health than the sum of their individual effects. This combination effect is called "synergism."

Since multiple pollutants commonly are present in polluted air, synergistic effects are a most important consideration. Photochemical pollutants also may contribute to the synergism and further increase the potential health hazard of the mixture. Laboratory experiments testing the effects of only one pollutant are mainly of academic interest, since polluted air usually contains a variety of contaminants.

Animal Studies

Because of its high solubility in water, a substantial amount of the sulfur dioxide inhaled through the nose will be absorbed by moisture in the nose and throat. At high ambient air concentrations (greater than 25 ppm) up to 98 percent of sulfur dioxide will be absorbed by mucous, but at lower levels more typical of ambient exposure (about 0.1 ppm) as much as 60 percent of the gas has been observed to reach the lungs of laboratory animals. Significant lung infiltration also occurs during mouth breathing (Strandberg, 1964; Amdur, 1966).

Even if the sulfur dioxide is removed from the air in the nose, it is rapidly absorbed into the blood and distributed throughout the body. Frank *et al*. (1967) severed the wind pipe of dogs and pumped radioactive sulfur dioxide through the nose and throat. Clean air was pumped into the lungs.

Most of the sulfur dioxide was absorbed in the nose and throat and exhaled from the lungs. This could only happen if the sulfur dioxide had been transported to the lungs through the dogs' blood. Radioactivity was measured in the blood within a few minutes after the start of the exposure and for several hours afterwards, indicating that sulfur dioxide had been absorbed. Radioactivity also was detected in the urine.

Once it enters the blood, sulfur dioxide reacts with proteins to form compounds (s-sulfonates) that may persist for several days after the exposure. It is possible that this is a protective mechanism rather than an adverse effect (Gunnison and Benton,1971). Sulfur dioxide also has been observed to inactivate certain enzymes and to interfere with cellular genetic material (DNA), but the significance of these effects is not clear.

Some experiments indicate sulfur dioxide may interact with basic cellular structures to facilitate or accelerate the onset of cancer in laboratory animals (Peacock and Spence, 1967; Laskin, 1970). Some epidemiological studies have suggested a correlation between lung cancer and sulfur dioxide or sulfate exposures (Higgins, 1974; Schwing and McDonald, 1976; Park _et al._, in press).

Numerous animal studies have confirmed that the lungs are highly sensitive to the sulfur dioxide and sulfate. Guinea pigs show statistically significant changes in lung function at sulfur dioxide concentrations as low as 0.16 ppm (Amdur, 1966). Microscopic examinations of lung tissue after exposure to sulfur dioxide have found many cells injured or killed and other cells abnormally increased in number. A combination of sulfur dioxide and fine carbon dust was found to injure cells and significantly increase the number of white blood cells in the deep lung at concentrations where either pollutant alone had no effect (Asmundson, _et al._, 1973).

In another experiment where neither sulfur dioxide nor inert particles alone had any effect on a group of guinea pigs, the two together were shown to significantly decrease the ability of natural protective mechanisms to defend against bacterial infection in the lungs (Rylander _et_ _al_.,1971).

Some particulate sulfates are substantially more irritating than an equivalent concentration of sulfur dioxide. The most irritating sulfur pollutant is a mist of sulfuric acid droplets. A 100 ug/m^3 mist of 0.3 micrometer sulfuric acid particles can increase breathing difficulty (airway resistance) in guinea pigs by 80 percent. An equal concentration of one micrometer sulfuric acid particles will only increase airway resistance by 10 percent, demonstrating that smaller particles have a greater effect. The sulfuric acid droplets are six to eight times more irritating than an equal weight of sulfur as sulfur dioxide (Amdur, 1978). A mist of 0.25 micrometer sulfuric acid particles at 30 ug/m^3 was found to alter lung function and to increase bacteria infection (Fairchild, 1975).

In earlier work several other sulfate compounds were shown to have an irritating effect like that of sulfuric acid. In particular, zinc ammonium sulfate particles were found to be about one-third and ferric sulfate particles about one-fourth as irritating as sulfuric acid mist of the same size and equivalent concentration. Ammonium sulfate has been shown to be about as irritating as sulfur dioxide (Amdur, 1978).

Administering these sulfate compounds along with sulfur dioxide previously had been found to result in synergistic effects. More recent work has led Amdur (1978) to attribute this effect to the formation of additional sulfuric acid by the metals in the sulfate compounds. Similarly, the sulfuric acid effect is implicated by McJilton _et_ _al_.(1973), who found that a relative humidity above 70 percent would significantly increase the adverse effect of a mixture of sulfur dioxide and sodium chloride (common table salt).

Clinical Studies

Sulfur dioxide has a characteristic, irritating odor which can be detected by most people at about 3 ppm. It is more easily detected by an "acidic" taste above about 0.3 ppm. Sulfuric acid mist can be detected by some people at 750 to 1000 ug/m^3. Most people will notice it at concentrations less than 3000 ug/m^3. Increased airway resistance has been observed during a 10 minute exposure at four to six ppm sulfur dioxide (Nadel et al., 1965). Similar results were observed with five minute exposures at 1 ppm to 3 ppm with breathing through the mouth (Lawther et al., 1975).

The reaction to sulfur dioxide exposures is quite variable. For example, two of ten subjects reacted quite strongly to a brief exposure of 5 ppm sulfur dioxide (Frank, 1962). In another study, three subjects suffered mild asthma attacks after a three-hour exposure to 0.5 ppm sulfur dioxide (Florida Sulfur Oxides Study, 1978). It has been estimated that about 10 to 20 percent of the population may be more than ordinarily sensitive to sulfur dioxide.

In order to observe the effects of lower concentrations of sulfur dioxide, several experiments have been performed using more sensitive tests of lung function. Tests measuring the effects on the small airways of the deep lung showed significant lung function impairment following exposure to one ppm of sulfur dioxide for 15 minutes (Snell and Luchsinger, 1969). Healthy young men who were engaged in light exercise registered a significant reduction in sensitive tests of function after 30 minutes exposure to 0.75 ppm sulfur dioxide (Bates and Hauzucha, 1973). Resting subjects registered marginally significant reductions in sensitive lung function tests after a 3-hour exposure to 0.5 ppm of sulfur dioxide (Florida Sulfur Oxides Study, 1978).

In another study designed to precisely measure the effects of sulfur dioxide Andersen et al. (1974) exposed 15 healthy young men to various sulfur dioxide levels and noted 1) discomfort of their subjects, 2) various lung function reductions, and 3) the rate of nasal mucous flow. It is important to note that the discomfort levels were elevated subjectively by the subjects themselves, rather than by changes in the pulse rate and other body functions that other studies have used in attempting to measure discomfort.

Mucous flow is believed to be an important part of the body's defenses against infection. At one ppm the mucous flow was reduced in sensitive individuals, and at five ppm the reduction was statistically significant for all subjects. Reduced mucous flow appeared to be closely related to the discomfort reported by the test subjects. At one ppm three of the subjects experienced slight discomfort. At five ppm eight subjects were uncomfortable and at 25 ppm eleven subjects registered discomfort. The degrees of discomfort reported by the subjects were found to correspond to the exposure levels. Impairment of lung function was statistically significant at all levels. During the week following the tests, a high rate of respiratory infections was observed among the subjects, although no cause and effect relation was established.

There is evidence that attempts to measure the joint effects of sulfur dioxide and other pollutants on human subjects may have measured primarily the effects of sulfuric acid. An exposure to 0.37 ppm of sulfur dioxide and 0.37 ppm of ozone for 30 minutes demonstrated substantial decreases in lung function for healthy young men engaged in an exercise-rest routine (Hauzucha and Bates, 1975). Bell et al. (1977) did not find as great an effect when they attempted to repeat the experiment. Careful analysis of the conflicting evidence suggests that there may have been a higher level of suspended particulates in the air used by Hauzucha and Bates, which could have contributed to the formation of fine sulfuric acid particles. Snell and Luchsinger (1969) observed a 12 percent decrease in lung function when young healthy subjects were exposed for 15 minutes to 0.5 ppm of sulfur dioxide and an estimated 1 ug/m^3 of 0.3 micron diameter distilled water mist. Toyama and Nakamura (1964) generated a sulfuric acid mist of 1.8 micron particles at 10 ug/m^3 to 100 ug/m^3 by mixing hydrogen peroxide and sulfur dioxide. They observed an 18 percent increase in airway resistance in their subjects.

For many people, some protection from sulfuric acid may result naturally from the generation of ammonia in the mouth. This would convert the sulfuric acid droplet to ammonium sulfate, which has been observed to be much less harmful. The amount of ammonia generated varies significantly from person to person (Larson et al., 1977).

Epidemiological Studies

Properly performed epidemiological studies are very valuable in identifying the relative importance of various pollutants in contributing to sickness and death. No sophisticated analysis was required to evaluate the effects of a smoke and fog blanket which settled over the Meuse Valley, Belgium, in the first week of December, 1930. By the end of the week several hundred people were seriously ill and 63 were dead. A similar incident occurred in the industrial valley around Donora, Pennsylvania, in October, 1948. It affected almost half the population and twenty persons died. No pollution measurements were made but sulfur dioxide, various sulfates, particulates, and even hydrogen fluoride have been suggested as possible causes of death and illness.

In December, 1952, the world's worst smog disaster occurred in London. Within a week more than 25,000 people had taken ill and 4,700 died. Measurements made in the most polluted areas indicated that the highest daily concentration of sulfur dioxide was about 1.3 ppm with about 4500 ug/m^3 of suspended particulate.

Episodes occurred in London again in 1956, 1957 and 1962. In each instance an increase in death rates was noted. Lawther (1963) analyzed the data relating to these episodes and concluded that increased death rates could be expected if sulfur dioxide exceeded 0.25 ppm and particulate exceeded 500 ug/m^3 measured by the British Smoke (BS) method, each on a daily average.

In an analysis of five smog episodes in New York City from 1962 to 1964, McCarroll and Bradley (1966) found a correspondence between death rates and pollution, even after statistical corrections for the effects of weather and influenza epidemics. While they did not identify threshold level where death rates would increase their results did not contradict Lawther's conclusions.

A study of a 1975 Pittsburg air pollution episode found fourteen deaths that could be attributed to pollution. During the time of the episode, daily average suspended particulate levels were about 450 ug/m^3 measured by the hi-vol (TSP) method, with a maximum particulate concentration of 1000 ug/m^3

TSP at one station. Suspended sulfate averaged 67 ug/m^3 TSP with a maximum of 266 ug/m^3 TSP at one station. The maximum 24-hour level of sulfur dioxide was 0.13 ppm (Riggan *et al*., 1975; Stebbings *et al*., 1976).

Changes in death rates do not clearly indicate the adverse health effects of air pollution since pollution is almost always only one of many contributors to the deaths. Nevertheless, numerous studies have used death statistics to determine the role of air pollution since they often are the only uniformly collected and accessible health related statistics. Two different techniques have been used. One compares variations of death rates in a community as pollution levels fluctuate over time. The other makes death rate comparisons between different communities with different levels of pollution over the same period of time (often as a yearly average).

Conflicting results have been obtained in studies of the daily death rates in the New York-New Jersey metropolitan area. Buechley *et al*. (1973) showed a correlation between an increase in daily death rates and increasing sulfur dioxide concentrations. No exposure threshold was found for the onset of increased death rates, but the relationship was more pronounced above 0.11 ppm and stronger still above 0.19 ppm (24-hour average). Particulates were included as a variable in the analysis and found to be only half as important as sulfur dioxide in increasing death rates. In a later analysis of New York City daily death rate data, Schimmel and Murawski (1976) found sulfur dioxide and particulate (measured as CoH) to be about equally important during the same years used by Buechley *et al*. Particulate was much more important during other years. As a result of this study Schimmel and Murawski argue that particulate or some other pollutant is responsible for the increased death rates and that "the SO_2 standards should either be greatly relaxed or abandoned entirely."

A strong correlation between daily levels of suspended particulate and daily death rates also was demonstrated by Lebowitz (1973) using data from New York, Philadelphia, and Los Angeles for the years 1962 to 1965.

A study of deaths caused by lung cancer and bronchitis in two neighborhoods of Eston, England, was carried out by Wicken and Buck (1964). After corrections were made for age, social class, smoking habits and occupational exposures, there remained a significant difference in the death rates, which the authors attributed to air pollution. The annual average pollution levels in the more polluted neighborhood were 0.04 ppm sulfur dioxide and 160 ug/m^3 BS of particulate. In the "clean" neighborhood the pollution levels were 0.03 ppm sulfur dioxide and 80 ug/m^3 BS particulates.

The Eston-type investigation can be extended to many cities. Schwing and McDonald (1976), Lave and Seskin (1977), and Mendelsohn and Orcutt (1978) have performed sophisticated analyses of annual death statistics and pollutant variables for urban areas in the United States. Schwing and McDonald compared seven pollutants (excluding particulate), six climate variables and ten social characteristics with death rates for white males in fifteen disease categories in 46 cities. Although cigarette consumption per capita almost always was the most important influence in determining the number of deaths for each disease class, sulfur dioxide and sulfate levels almost always were the second and third most important. The associations with sulfur dioxide and sulfate were particularly strong for deaths caused by lung cancer and heart diseases. Lave and Seskin compared levels of particulates, sulfur dioxide and sulfate and four socio-economic variables with total deaths in up to 117 urban areas. Statistically significant associations were found between the air pollution variations and death rates. In their research on pollutant effects, Mendelsohn and Orcutt used 28 personal characteristics, four climate variables, 400 urban areas, and seven pollutants, including sulfur dioxide, sulfates and particulate. Deaths from illness were divided into five general groups. Both sulfate and particulate levels showed a strong association with deaths from heart disease and sulfur dioxide was associated with deaths from lung disease.

In a major long-term study on the health of children, British researchers collected data on about 4,000 children all born the same week fifteen years earlier. The children were divided into four groups according to the extent of pollution at their place of residence. Sickness due to infections of the lower lungs and absences from school due to bronchitis increased as the level of pollution increased. There was a high level of statistical significance. Although children of wealthier families might receive better medical care, the results were not affected by the social class of the parents. Even the children in the least polluted area may have showed some pollution effects but this could not be known because there was no cleaner area in the study for comparison of effects. Effects could be clearly observed in the second least polluted area by comparing them with those from the least polluted area. Annual average concentrations in the second least polluted area were estimated to be 0.05 ppm sulfur dioxide and 132 ug/m^3 BS suspended particulate. (Douglas and Waller, 1966)

In Sheffield, England, the topography of the land and the siting of the industry results in a substantial variation of pollution across the city. Thus it was possible in another experiment to study the children in five elementary schools and assign them to four different classes of pollutant exposure. Health statistics and socio-economic information were collected on 800 children. The children were examined by a physician and lung function tests administered. Even after adjusting for parent's social class, there was a consistent and statistically significant increase in both upper and lower respiratory tract infections as the pollution level increased. Impaired lung function also was observed in children with a history of respiratory disease. No threshold was observed, but the pollution levels for the second least polluted area were 0.07 ppm sulfur dioxide (annual average) and about 230 ug/m^3 BS particulate (annual average) (Lunn, et al., 1967)

Perhaps the most extensive study of air pollution-related health effects was the postal survey by Lambert and Reid (1970). Nearly 10,000 British adults (74 percent of the sample) responded to a detailed questionnaire on their heart and lung health status and personal information, such as smoking habits and occupation. Again, it was possible to divide the subjects into groups corresponding to four areas with differing pollution levels. Lambert and Reid found a persistent increase in the incidence of symptoms (phlegm produced by a cough) and chronic bronchitis with each increase in air pollution level. When the smokers and nonsmokers in the sample were analyzed separately it was found that the symptoms and chronic bronchitis rates increased much more sharply for the smokers. Lambert and Reid concluded,

> This widening gap between smokers and nonsmokers as pollution increases certainly suggests that the joint influence of pollution and smoking on the level of the prevalence-rate is greater than the sum of their separate effects.

The importance of the "productive cough" symptom was underscored by a follow-up survey which found that those who reported these symptoms had a nine-fold higher death rate from chronic lung diseases than those not reporting them.

An analysis of work absences due to illness at a large New York insurance office provided a unique opportunity to determine the specific cause of such absences. When the absence rates were compared to pollution levels a significant correlation was found between the number of absences caused by respiratory ailments and levels of sulfur dioxide and particulate (CoH). No correlation was found for nonrespiratory illness absences. A clear parallel between work absences and both sulfur dioxide and particulate levels could be seen over the range from the lowest to the highest pollutant levels. When mathematical models were constructed to explain variabilities in the trend about 50 percent of the absences were found to be related to an annual seasonal temperature variation. Pollution levels could account for up to 10 percent of the absences. Most of the pollution related absences occurred on days with particulate levels above 1.6 CoH (Verma, 1969).

A recent study of emergency room admissions at a Chicago hospital also demonstrated a significant correlation between health and air pollution. In this instance, sulfur dioxide was found to be the most significant pollutant (Fishelson and Graves, 1978). The average sulfur dioxide level recorded during the study was 0.025 ppm (Graves, personal communication, 1978).

In order to help separate the effects of particulates from those of sulfur dioxide, one study team sought out two cities with very low levels of sulfur dioxide, one of which had high particulate levels and the other low. The cities chosen were Charlotte, North Carolina with low particulate levels and Birmingham, Alabama with high particulate levels. The measured pollutant levels in these cities are shown in Table III.A.-1. Approximately 5,000 families completed questionnaires on the respiratory disease history of their children. Children with a history of asthma were excluded. Among white children in Birmingham, there was a significantly higher incidence of croup, bronchitis, pneumonia, and lower respiratory disease. Among black children the same differences were observed, except with bronchitis. The relative risk of childhood lower respiratory disease appeared to be 1.2 to 2 times greater in Birmingham than in Charlotte (Hammer, 1977).

TABLE III.A.-1 Pollutant Levels by City (ug/m^3 TSP)

		Charlotte	Birmingham
Total Suspended Particulates	1972	70	127
	1967-1972 avg.	78	138
Particulates smaller than 3.5 micrometers	1972	39	55
	1967-1972 avg.	43	56
Suspended sulfates	1972	9.7	13.3
	1967-1972 avg.	9.5	12.3
Sulfur dioxide	1972	0.006 ppm	0.004 ppm
	1967-1972 avg.	0.006 ppm	0.005 ppm

Source: Chapman *et al.*, 1976

Sensitive Groups

In an attempt to find more sensitive pollution indicators than the illness rates of generally healthy populations, some researchers have tested lung function, examined hospital patients, children, and persons with chronic lung diseases or asthma. They have generally observed effects at lower levels than were evident in the illness studies of the general population.

Van Der Lende *et al.* (1975) measured lung function in almost 4,000 men and women in industrial and rural Netherlands communities in 1969 and 1972. Although lung function normally declines with age, repeat measurements found significant lung function improvement in the industrial community, particularly among persons 25 to 39 years of age. This was attributed to the decline in air pollution. During the 1969 testing the average 24-hour sulfur dioxide levels were 0.08 ppm and the average suspended particulate levels were 88 ug/m^3 BS. In 1972 the levels were 0.03 ppm and 25 ug/m^3 BS, respectively. About 16 percent of the 25 to 39 age-group showed an improvement greater than 10 percent and 41 percent showed an improvement of 10 percent or less. There tended to be a slightly higher incidence of chronic lung disease among those who had been helped the most by the improvement in air quality, indicating a possible greater sensitivity among this part of the population.

Lung function was measured in two studies of school children who experienced different levels of pollution. As a part of the Charlotte-Birmingham study mentioned earlier, 8,000 children were tested in elementary schools in the two cities. More than 5000 children were tested three times, once each in fall, winter and spring. The children in Birmingham showed highly significant decreases in lung function, corresponding to higher particulate levels. The authors attribute the lung function decrease to the increased total suspended particulate levels in Birmingham, but suggest that the fine particulate may be the more specific cause (Chapman _et_ _al_., 1976).

A similar study in Cincinnati tested 400 elementary school children in the fall, winter and spring. Significant differences in measured lung functions were observed between children from the less polluted sections of the city and those from more polluted sections. The subjects were grouped according to family income and race and tested for lung function. When the results were compared with pollution levels, the pollutant appearing to have the greatest effect was suspended sulfate. Total suspended particulate had no significant effect on performance. It is not possible in this study to identify the daily levels of suspended sulfate necessary to cause decrease in lung function (Shy *et al.*, 1978).

A study of hospital patients attempted to determine the correspondence between the length of their stay and daily pollution levels during their hospitalization. More than 30,000 admission records from all large hospitals near an air pollution monitoring station in Los Angeles were analyzed over an eight-month period. Sulfur dioxide levels were found to be the most consistent influence on lengthened periods of care, particularly for patients with heart and lung diseases. Particulate and ozone also were important for these diseases. Nitrogen oxides related strongly to infections. Pollutant levels were relatively stable during the period of the study, with no levels above the normal for the area. All pollutant levels were averaged over 24 hours, except ozone. Ozone was read hourly each day, and the highest daily readings were averaged over the test period. The average concentrations were: for ozone, 0.15 ppm; for particulate, 2.9 KM units; and for sulfur dioxide, 0.01 ppm. This sulfur dioxide level is far below any other reported to cause measured effects (Sterling, 1967).

Two studies of the health status of elderly bronchitis patients have found parallels between increased air pollution and illness. Lawther *et al.* (1970) studied 1,000 individuals living in London in 1959-60 and again in 1964-65. Another sample of 50 individuals was studied in 1967-68. Each person was asked to record daily if his chest condition was better, the same, or worse than the previous day. These reports were averaged and compared to pollution levels. A remarkably correlated response to pollutant levels was observed. Between 1960 and 1964 suspended particulate was reduced, but sulfur dioxide levels stayed the same. Because of differences in the weather, a strict comparison between the earlier and later studies could not be made, but the authors noted,

> When the two winters were compared in terms of the association with sulfur dioxide, the general impression was of a slightly reduced and less consistent response in 1964-65 as compared with 1959-60. It was clear, however, that some association remained which had not been reduced in proportion to the reduction in smoke concentration.

The correlations between the pollutant levels and the health status were much weaker in 1965-65. From these two study periods, Lawther concluded that the minimum daily pollution level which would generate a significant response among bronchitis patients was about 0.19 ppm of sulfur dioxide and about 250 ug/m^3 BS of particulate. When the study was repeated in 1967-68 the same correlations were seen, but there was only one day when the pollution levels exceeded those previously believed to be the threshold level for the effect. Suspended particulate, sulfur dioxide and sulfuric acid mist were all significant and approximately equally important in eliciting the effect. The average winter levels in 1967-68 were: sulfur dioxide, 0.08 ppm; particulate, 204 ug/m^3 BS; sulfuric acid, 6.3 ug/m^3.

The 1966-67 Chicago study included 560 bronchitis patients and used techniques similar to those used in the London studies. Air pollution monitoring stations near each individual's residence and place of work were used to determine a total exposure to sulfur dioxide. Effects of other pollutants were not evaluated. The degree of illness in the group of advanced bronchitis patients over 55 years of age was found to increase uniformly with each level of pollution increase from 0.04 ppm to above 0.3 ppm (24-hour average). Younger patients with advanced bronchitis showed a clear response to pollution levels above 0.25 ppm (24-hour average). The average levels of suspended particulates during the study period was 120 ug/m^3 TSP (Carnow _et al_., 1969).

Some individuals with asthma are more sensitive to air pollution than the general population is. Therefore, persons with asthma often are selected for special study. In October of 1963 a concerted effort was made to find the cause of recurring "epidemics" of asthma in New Orleans. Numerous air monitoring devices not commonly used were available to characterize the pollution. Kenline (1966) used sophisticated particle counting devices to show that the concentration of particles less than eight micrometers diameter, and more specifically, particles less than one micrometer diameter, corresponded very closely to the number of persons reporting asthma complaints to a hospital emergency room. The complaints were at a maximum on the day when the particles less than eight microns reached 95 ug/m^3. Kenline used other measurements to show that there was no correlation between asthma complaints and levels of particulates, organic compounds, pollen or spores.

In another investigation, a group of twenty asthma patients living near a coal-fired power plant was studied for seven months. The patients were asked to keep diaries of asthma attacks, which later were compared to measured air pollution levels. During three weeks of the study the subjects were examined by a doctor to confirm the self diagnosis of an attack. No threshold was found, with attack rates increasing uniformly from the lowest levels of pollution to the highest. When corrections were made

TABLE III.A-II SUMMARY: HEALTH EFFECTS OF SULFUR OXIDES AND PARTICULATE MATTER

Subjects	Pollution/ Concentration	Exposure Time	Observed Effect	Reference
Normal males	5 ppm SO_2	10 minutes	Two subjects had severe coughing and wheezing	Frank et al., 1962
Normal males	1 ppm SO_2	15 minutes	Decrease in lung function	Snell and Luschinger 1969
Exercising males	0.75 ppm SO_2	30 minutes	Decrease in lung function	Bates and Hauzucha, 1973
Exercising males	0.37 ppm SO_2 + 0.37 ppm ozone + particles (?)	30 minutes	Decrease in lung function	Hauzucha and Bates, 1975
Normal males	1.8 micron sulfuric acid mist 10 ug/m^3	5 minutes	Increased airway resistance	Toyama and Nakamura, 1964
General population	0.04 ppm SO_2 160 ug/m^3 BS	annual annual	Increased death rates from lung cancer and bronchitis	Wicken and Buck, 1964
Children	0.07 ppm SO_2 230 ug/m^3 BS	annual annual	No observed threshold for increased respiratory disease	Lunn, 1967
General population	0.04 ppm SO_2 100 ug/m^3 BS	annual	No observed threshold for increased respiratory disease	Lambert and Reid, 1970
Children	0.01 ppm SO_2 138 ug/m^3 TSP suspended sulfates 12 ug/m^3 TSP	annual annual annual	Increased respiratory disease	Hammer, 1977
General population	0.08 ppm SO_2 88 ug/m^3 BS	24-hour 24-hour	Decrease in lung function	Van der Lende et al., 1975
Children	0.01 ppm SO_2 127 ug/m^3 TSP suspended sulfates 13 ug/m^3 TSP	annual annual annual	Decrease in lung function	Chapman, et al., 1976
Elderly bronchitis patients	0.08 ppm SO_2 204 ug/m^3 BS sulfuric acid 6.3 ug/m^3	24 hours 24 hours 24 hours	No observed threshold for increased bronchitis symptoms	Lawther et al., 1970
Asthma patients	particles less than 8 microns 95 ug/m^3	24 hours	Substantial increase in visits to emergency room	Kenline, 1966
Asthma patients	0.07 ppm SO_2 150 ug/m^3	24 hours	No observed threshold of increased rate of asthma attack	Cohen et al., 1972

SO_2: sulfur dioxide BS: particulate by British Smoke method TSP: particulates by hi-vol method for temperature effects, the strongest influence on attack rates was suspended sulfates, although sulfur dioxide, AISI readings, and total suspended particulate levels also were important. It was possible to define average levels of pollutants which divided "low" and "high" attack days. The dividing line was 0.07 ppm sulfur dioxide and 150 ug/m^3 total suspended particulate (Cohen, 1972).

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Effects of Sulfur Dioxide on Crops and Other Plants

Recent work by plant scientists shows sulfur dioxide can injure vegetation at much lower levels than previously thought, especially when sulfur dioxide is combined with other pollutants as often occurs in the atmosphere. Experiments show that sulfur dioxide at low levels can act synergistically with other pollutants to cause plant damage more severe than when sulfur dioxide acts alone. Among the pollutants acting with sulfur dioxide to produce such damage are ozone, nitrogen oxides, peroxyacetylnitrates, particulates, fluorides, and hydrochloric acid (Guderian, 1977). Ashenden *et al*. (1977) aptly summarize:

> It is not, however, of any practical value to consider the effects of these gases separately when it is known they can act additively on net photosynthesis, and synergistically on the activity of some important enzymes, and in producing externally visible injury. For this reason, it is unwise to regard a pollutant, for example sulfur dioxide, as being present at a concentration too low to exert an effect.

Table IIIA-III lists the adverse vegetation effects caused by sulfur dioxide (at a concentration of 0.5 ppm or less) alone and in combination with other pollutants found in Montana. The table is arranged by duration of exposure, with effects from short-term exposures presented first, followed by effects resulting from continued or repeated exposures over longer periods. The results of these experiments range from subtle changes in plant function at short exposures to reduced growth and yield, accelerated aging, delayed development of plant organs, leaf destruction, and pollen abnormalities at long exposures. Table IIIA-III indicates that a number of Montana's natural and agricultural plant species are sensitive to sulfur dioxide either alone or in combination with other pollutants. Additional plants which are

TABLE III. A-III SUMMARY: EFFECTS OF SULFUR DIOXIDE ALONE AND IN COMBINATION WITH OTHER POLLUTANTS TO SELECTED PLANTS AT 0.50 PPM OR LESS

Plant Species	Concentration	Exposure	Response of Vegetation	References
Bean 4-pair leaf and 5-pair leaf stages	0.05 ppm Sulfur Dioxide (SO_2)	10 min.	Stomata opened wider (measured as decreased diffusive resistance); A similar response occurred at 0.10 ppm and 0.45 ppm. Oldest leaves were most sensitive (stomatal resistance increased with leaf age irrespective of the SO_2 concentrations).	Biscoe et al. 1973
Bean	0.02 ppm SO_2	10 min.	Stomata opened wider (measured as decreased diffusive resistance).	Unsworth et al. 1972
Corn at 4-leaf state	0.01 ppm SO_2	10 min.	Stomata opened wider (measured as decreased diffusive resistance).	
Red pine seedlings	0.5 ppm SO_2	15 min.	Reduction in chlorophyll in primary needles Decrease in dry weight (growth) of cotyledons and primary needles Results were greatly magnified when exposure was continued for 30, 60, and 120 minutes.	Constantindou et al. 1976
Beans and Corn	0.10 ppm SO_2	20 min.	Stomata opened wider (measured as decreased diffusive resistance). No difference between plants exposed in dark or light: SO_2 opens stomates in the dark	Unsworth et al. 1972
Pea	0.05 ppm SO_2 + 0.05 ppm Nitrogen Dioxide (NO_2)	1 hour	Significantly decreased net photosynthesis	Bull & Mansfield, 1974
Beans 4-leaf stage	0.10 ppm SO_2	1 hour	Stomata open wider (measured as decreased diffusive resistance). Note: Water-stressed plants exhibited wider opening and an earlier response than fully watered plants.	Unsworth et al. 1972
Alfalfa	0.15 ppm SO_2 + 0.15 ppm NO_x	1 hour	Inhibits photosynthesis, percent of reduction not stated, but authors state synergistic effect most marked at this concentration.	White et al. 1974
Alfalfa	0.15 SO_2 + 0.15 ppm NO_2	1 hour	Greater-than-additive inhibition (7%) photosynthesis (as measured by CO_2 uptake). Similar results at 0.25 ppm for both gases.	Bennett & Hill, 1974
Alfalfa	0.35 ppm SO_2	1 hour	8% reduction in apparent photosynthesis	White et al. 1974
Spongy mesophyll of 25-day old broad bean leaves at second node from base.	0.25 ppm SO_2	1 hour	Slight swelling of stroma thylakoids of chloroplasts. Author states implication is impairment of photosynthesis. At 2 hrs. at 0.25 ppm swelling spread to granum thylakoids. These effects reversible.	Wellburn et al. 1972
Alfalfa	0.30 ppm SO_2 + 0.10 ppm Ozone (O_3)	1 hour	Additive inhibition of photosynthesis 11% ± 3% measured by CO_2 uptake. Some tissue damage.	Bennett & Hill, 1974
Oats	0.50 ppm SO_2	1.5 hour	Initiation of leaf injury	Heck & Dunning, 1976
Eastern white pine	0.05 ppm SO_2	2 hour	Caused tip damage on new needles	Costonis, 1973

Plant Species	Concentration	Exposure	Response of Vegetation	References
Alfalfa	0.15 ppm SO_2 + 0.15 ppm NO_2	2 hours	7% reduction in apparent photosynthesis. Some tissue death	White et al. 1974
Oat, Barley, Alfalfa	0.2 ppm SO_2	2 hours	Photosynthesis inhibition begins (as measured by CO_2 uptake rate compared to controls).	Bennett and Hill, 1974
Eastern White Pine	0.25 ppm SO_2	2 hours	6.5% of exposed parts injured. Needle damage.	Berry, 1971
Jack Pine	0.25 ppm SO_2	2 hours	4.5% of exposed parts injured. Needle damage.	Berry, 1971
Peanut	0.02 to 0.03 ppm SO_2	2-48 hours	Plants fumigated until leaf markings first appeared at all concentrations, the symptoms appeared on high moisture plants first, followed in 8 to 10 hours on medium moisture, and 10 to 14 hours on low-moisture.	Applegate and Durrant, 1969
Low moisture = wilted, then enough water for limited growth	0.02 to 0.03 ppm SO_2 + 0.008 to 0.01 O_3	2-3 hours	First symtom: O_3 - type injury on youngest leaves, 46 to 56 hours of fumigation later turned color similar to SO_2 damage.	
Medium moisture = normal watering	0.02 to 0.12 ppm SO_2	4-8 hours	First symptom: Mature leaves lost chlorophyll. Entire leaf lost when tissue death complete.	
High moisture = grown in beakers of water	0.02 to 0.03 ppm SO_2	24-48 hours	First symptom: Mature leaves lost chlorophyll and fell off plant. Did not show tissue death.	
Oat	0.40 ppm SO_2	3 hours	Threshold for leaf injury	Heck and Dunning, 1976
Radish	0.05 ppm SO_2 + 0.10 ppm NO_2	4 hours	17% leaf injury, lower leaf surface 0.5 ppm SO_2 alone = threshold; 2 ppm O_3 alone = threshold.	Tingey et al. 1971
Pinto Bean	0.05 ppm SO_2 + 0.05 ppm NO_2	4 hours	2% leaf injury measured as pigmented lesion, tissue death, loss of chlorophyll.	Tingey et al. 1971
Radish	"	4 hours	tissue death and loss of chlorophyll	
Oats	"	4 hours	tissue death and loss of chlorophyll	
Eastern White Pine	0.05 ppm SO_2 + 0.05 ppm O_3	4 hours	Severe needle injury (2-3 cm leaf tip injured) on new needles, expressed most vividly 72 hours after exposure.	Costonis, 1973
Radish	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	50% leaf injury	Tingey et al. 1973
Cabbage	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	22% leaf injury. 0.10 to 0.5 ppm SO_2 alone = no foliar injury; 0.05 ppm O_3 alone = 1% foliar injury	Tingey et al. 1973
Broccoli	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	34% leaf injury. 0.05 ppm SO_2 alone = 4% leaf injury; 0.10 ppm O_3 alone = 1% leaf injury.	Tingey et al. 1973
Tomato	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	50% leaf injury	Tingey et al. 1973
Radish	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	50% leaf injury	Tingey et al. 1973
Alfalfa	0.10 ppm SO_2 + 0.10 ppm O_3	4 hours	24% leaf injury	Tingey et al. 1973
Bel B. Tobacco	0.25 ppm SO_2	4 hours	1% leaf injury	Tingey et al. 1973

Plant Species	Concentration	Exposure	Response of Vegetation	References
Cottonwood	0.3 ppm SO_2	4 hours	Significant decreases in moist pollen tube length	Karnowsky et al. 1974
Broccoli	0.50 ppm SO_2	4 hours	4% leaf injury expressed as the precentage of leaf surface.	Tingey et al.1973
Alfalfa	0.50 ppm SO_2	4 hours	19% leaf injury	Tingey et al.1973
Cabbage	0.50 ppm SO_2 + 0.05 ppm O_3	4 hours	4% leaf injury. 0.10 ppm to 0.5 ppm SO_2 alone = no leaf injury; 0.05 ppm O_3 alone = no leaf injury	Tingey et al.1973
Broccoli	0.50 ppm SO_2 + 0.05 ppm O_3	4 hours 4 hours	17% leaf injury. 0.5 ppm SO_2 alone = 4% leaf injury 0.10 ppm O_3 alone = 1% leaf injury.	Tingey et al.1973
Radish	0.50 ppm SO_2 + 0.05 ppm O_3	4 hours	7% leaf injury	Tingey et al.1973
Peas	0.10 ppm SO_2 + 0.10 ppm O_3	5 hours	14.6% damage. Leaf surface destruction more severe on lower leaves. Ash colored spots on intermediate leaves, and veins.	Fujiwara et al.1973
Peas	0.05 ppm SO_2 + 0.10 ppm O_3	5 hours	9% visible injury. Upper leaves showed ozone injury; lower leaves showed O_3 leaf surface destruction. Author calls this response synergy.	Fujiwara et al.1973
	0.10 ppm SO_2 + 0.10 ppm O_3	5 hours	14.6% leaf tissue death	
Eastern White Pine	0.025 ppm SO_2	6 hours	Threshold for needle damage. Response judged in terms of needle elongation (growth) and needle lesions or tip damage. 0.025 SO_2 + 0.05 ozone showed significant response to all sensitive plants.	Houston, 1974
Buckwheat 8-10 leaf state	0.46 ppm SO_2	7 hours	Slight injury to a few leaves.	Zimmerman and Crocker, 1934
Tobacco	0.10 ppm SO_2	8 hours	Threshold for fleck like lesions on leaves.	MacDowall and Cole, 1971
Tradescantia	0.074 ppm SO_2	18 hours	Increased chromosone aberration rate of germinating pollen	Ma et al.1973
Lodgepole Pine	100 to 500 ppm aqueous = 0.1 to 0.5 ppm in the air.	22 hours	Disruption of chloroplasts and reduction in photosynthesis	Malhotra, 1976
Douglas Fir seedlings	0.29 ppm SO_2	44 hours	Visible injury	Katz and McCallum, 1939
Oats	0.50 ppm SO_2	4 hours/day for 14 days (56 hrs)	Head number unchanged. Top dry Carolee weight reduced 3%. Root dry weight reduced 13%. Greenhouse grown, Exposure chamber.	Heck et al.1975
Peas	0.15 ppm SO_2 + 0.10 NO_2	6 days 24 hrs/day (144 hrs)	15% reduction in RDP enzymes. Synergism at metabolic levels	Horsman and Welburn, 1975
Loblolly Pine (2 Wks. old) Sycamore (1 Wk. old) Seedlings	0.14 ppm SO_2 + 0.05 ppm O_3 + 0.10 ppm NO_2	6 hour/day for 28 days (168 hrs)	Significant growth reduction (measured as height) compared to either ozone and sulfur dioxide combined, or ozone alone. Needles were significantly shorter than for any other exposure. This study is an example of growth reduction with slight leaf symptoms. Leaf symtoms response most sensitive in early July.	Kress and Skelly, 1977

Plant Species	Concentration	Exposure	Response of Vegatation	References
Tomato	0.20 ppm SO_2	15 days	First sign of tissue death. By this time the vitamin content has been substantially reduced or changed (B_1, B_6 Nicotinic acid.)	Unzicker et al. 1975
Lettuce	0.29 ppm SO_2	15 days	Damage leaf areas, 30% thiamine content reduction	Unzicker et al. 1975
Peas	0.1 ppm SO_2	18 days	Significant reductions in protein and yield. Less significant loss of other nutrients	Jager and Klein, 1977
Orchard Grass	0.11 ppm SO_2	4 weeks	34% reduction in leaf size, 45% reduction in dry weight	Ashenden, 1978
Ryegrass	0.11 ppm SO_2	4 weeks	20% reduction in leaf size, 40% reduction in total dry weight	Ashenden and Mansfield, 1977
Ricegrass	0.06 ppm SO_2	6 weeks	Reductions in photosynthesis, respiration, and the chlorophyll content. Slight increase in productivity.	Ferenbaugh, 1978
	0.13 ppm SO_2	6 weeks	Decrease in productivity	
Duckweed	0.15 ppm SO_2	6 weeks (1008 hrs)	Reduction in size of fronds; starch content reduced.	Fankhauser et al. 1976
Duckweed	0.15 ppm SO_2	51 days (1224 hrs)	Reduction in starch, reduction in growth (measured as reduced surface area, average dry weight, reduced doubling time)	Fankhauser et al. 1976
Ryegrass	0.12 ppm SO_2	8 hour/day, 9 weeks (504 hrs) 8 hour/day, 26 weeks (1456 hrs)	46% redcution in yield measured as leaf and tiller production; increased loss of leaves, 52% reduction in dry shoot weight.	Bell and Clough, 1973
Ryegrass	0.073	26 weeks (4368 hrs)	50% reduction in dry weight of shoots	Bell and Mudd, 1976
Ryegrass	0.037	periodic harvest between 6 and 43 wks.	Whole plant dry weight of ryegrass grown either on a nutrient-rich sand culture or on unfertilized field soil was depressed for up to 36%	Crittenden and Read, 1978
A Number of Montana range plants.	SO_2 concentrations ranging from 0.02 ppm, 0.05 ppm, 0.15 ppm, 0.25 ppm	growing season	Few responses during 1st year of exposure A number of subtle effects at second year including: Earlier leaf fall, insect exodus, reduced species diversity, root/shoot impairments, possible seed germination delay, inhibition of litter decomposition.	
Scotch Pine, Norway Spruce, Douglas Fir, Frazier Fir	less than 0.01 ppm SO_2 0.01 ppm NO_2 0.07 ppm HF 0.05 ppm O_3 four month average during growing season	growing season	Growth abnormalities, needle loss and tissue death	EPA, 1971
Wheat	0.015 ppm SO_2	growing season	15 percent reduction in grain weight yield.	Guderian, 1977
Oats	0.01 ppm SO_2	growing season	leaf injury	Guderian, 1977
White Birch	0.006 ppm	growing season	No effects on sulfur content of foliage development of leaf SO_2 injury	Linzon, 1978
	0.011 ppm	growing season	Elevated sulfur levels trace to light leaf SO_2 injury	
	0.026 ppm	growing season	Moderate to severe leaf SO_2 injury	
	0.048 ppm	growing season	Severe SO_2 injury symptoms and leaf sulfur concentrations 3 times normal	

Plant Species	Concentration	Exposure	Response of Vegetation	References
Jack Pine	0.045 ppm	growing season	Injury to Jack Pine trees was a mixture of chlorophyll loss and tissue death	Linzon, 1978
Birch and Aspen foliage	0.008 ppm	growing season	Very slight injury 64 km NE of SO_2 source	
Lichens	0.041 ppm plus dust @ 5 g/m^3 ambient	monthly	Lichens completely absent.	Kirschbaum, 1971
Lichens	0.042 - 0.030	6 months	tissue death	LeBlanc *et al*. 1972
	0.006 - 0.018	6 months	loss chlorophyll and growth reduction	LeBlanc and Rao, 1973
Bryophytes	0.017	6 months (winter)	elimination of plants	Gilbert, 1968
Fir Forests	0.007 - 0.01 ppm	Annual average	20 ± 5% loss in growth. Premature drop of some needles, and reduction of above-growth plant organs.	Materna, 1973
Fir Forests	0.026- 0.037 ppm	Annual average	Rapid death of whole groups of trees.	
Lichens	0.01 - 0.02	annual	Elimination of many lichen species	LeBlanc *et al*.1972
Lichens	Below 0.011 ppm	long-term	No effect.	Hawksworth, 1973

not grown in the state but are closely related to Montana species and which have been the subject of extensive experimentation are also included this table.

Changes in Plant Function

Subtle changes in plant functioning are among the earliest signs of sulfur dioxide effects. It is generally accepted that sulfur dioxide injury depends on entry through pores (stomata) on the leaf surface. Stomata regulate gas exchange and water loss in plants by varying the diameter of their opening. One of the first indicators of sulfur dioxide stress in plants is disruption in the normal operation of the stomata. Unsworth et al. (1972) have shown that a ten minute exposure to as little as 0.01 ppm sulfur dioxide causes stomata to increase in diameter, while 0.1 ppm sulfur dioxide causes stomata to open abnormally in the dark. Malfunctions of stomata have three undesirable consequences in plants: increased water loss during dry weather, increased plant uptake of pollutants, and increased fungal infections. Rosen et al. (1978) point out that the relationship of sulfur dioxide to stomatal malfunction and injury is complex:

> Under varying conditions, changes in stomatal resistance were not sufficient to account for changes in injury. Other studies have shown a negative correlation between injury and stomatal resistance, indicating that injury is a consequence of increased stomatal aperature. Conversely, stomatal aperature (changes) has been shown to be a consequence of injury. (Citations omitted)

Sulfur is an essential constituent of many enzymes necessary for normal plant growth. Normally, the plant receives adequate sulfur from the soil but can utilize sulfur dioxide absorbed through the stomata to fulfill nutritional requirements (Thomas et al., 1943). Plant sensitivity appears related to its ability to digest (metabolize) sulfur dioxide. This ability depends on the environmental conditions under which the plant is grown and varies in degree both in plants of the same species and between species (Gudərian, 1977). McCool and Johnson (1938) reported that sensitive species of ragweed accumulated less sulfur before the onset of injury than resistant species.

Plant response to sulfur dioxide is generally related to the concentration of pollutant multiplied by the exposure duration (dosage)(O'Gara, 1922). This means that a plant can be injured as much by a short-term high concentration as by a long-term low concentration of sulfur dioxide. A number of researchers have used total sulfur in foliage as an indicator of sulfur dioxide dosage and plant stress (EPA, 1978; Linzon, 1978).

When the dosage of sulfur dioxide accumulates above a level the plant can tolerate, chemical changes take place that inhibit photosynthesis and respiration (Malhotra, 1976), decrease chlorophyll content (Rao and Le Blanc, 1966) and cause reductions in essential enzymes (Mukerji and Yang, 1974). These disruptions can cause growth and yield losses without visible injury (Thomas *et al*., 1961). Plant scientists agree that sulfur dioxide induced internal disruptions in plant processes, if severe enough, will cause the onset of visible injury either through tissue death or loss of chlorophyll in the leaf (Mudd, 1975). Entire plant populations have died from exposure to high levels of sulfur dioxide (Scheffer and Hedgecock, 1955).

Dose-Response Effects on Agricultural Plants

Alfalfa is an important agricultural crop in Montana. A number of independent field and laboratory studies have found that alfalfa is highly sensitive to sulfur dioxide in comparison to other plant species (Davis and

Wilhour, 1976). Experiments by White et al. (1974) and Bennett and Hill (1974) show that 0.35 ppm sulfur dioxide for one hour and 0.20 ppm for two hours caused a measurable decrease in photosynthesis. Tingey et al. (1973) noted that a 0.5 ppm sulfur dioxide exposure level continued for four hours caused 19 percent leaf injury.

When combined with other commonly occurring pollutants, sulfur dioxide toxicity to alfalfa is heightened. White et al. (1974) and Bennett and Hill (1974) found that a one hour exposure to 0.15 ppm sulfur dioxide combined with 0.15 ppm nitrogen dioxide caused a seven percent reduction in photosynthesis. Similar results were noted by Bennett and Hill (1974) after a one hour exposure with 0.3 ppm sulfur dioxide and 0.1 ppm ozone. A four hour exposure with 0.1 ppm sulfur dioxide and 0.1 ppm ozone was found by Tingey et al. (1973) to cause a 24 percent leaf injury in alfalfa.

Other agricultural plants sensitive to sulfur dioxide are wheat, oats, and barley. Experiments shown in Table III.A-III indicate reductions in photosynthesis occur at 0.2 ppm sulfur dioxide after a one hour exposure for barley and oats (Bennett and Hill, 1974), with the beginning of leaf injury in oats at 0.4 ppm sulfur dioxide after three hours (Heck and Dunning, 1976). Guderian (1977) noted that a growing season average of 0.015 ppm sulfur dioxide decreased grain weight yield in wheat by 15 percent. He also found that an average sulfur dioxide level of 0.01 ppm for the growing season caused slight leaf injury to oats.

Truck and home garden vegetables such as radishes, tomatoes, broccoli, and cabbage show significant visible injury after a four hour exposure to a combination of 0.5 ppm sulfur dioxide and 0.05 ppm ozone. With 15 day exposures, tomato shows leaf injury from 0.20 ppm sulfur dioxide and lettuce from 0.29 ppm (Unzicker et al., 1975). Peas were found to be sensitive to sulfur dioxide alone and in combination with ozone. Jager and Klein (1977) found that an 18 day exposure of peas to 0.1 ppm sulfur dioxide caused significant yield and protein reductions. The only visible signs of injury at this concentration was a slight yellowing from a loss in chlorophyll. Fugiwara *et al.* (1973) showed that 0.05 ppm sulfur dioxide with 0.1 ppm ozone caused synergistic leaf damage after five hours of exposure.

Dose-Response Effects on Grasslands

Several experiments have assessed the impact of sulfur dioxide on range grass ecosystems. The EPA is cooperating with several western universities in studying the effects of long-term low concentration sulfur dioxide exposures on eastern Montana rangelands near Colstrip, Montana. Research results from Colstrip describe visible injury to western wheatgrass with annual exposure levels of sulfur dioxide between 0.02 and 0.10 ppm. There appeared to be a delay in initial growth of plant parts both above and below ground surface. Results also showed delays in the distribution of plant nutrients from leaves to the roots and an accelerated loss of older leaves. The decomposition of dead plant material was slowed by annual sulfur dioxide concentrations between 0.02 and 0.10 ppm. A buildup of such material, although possibly decreasing erosion rates, also would decrease soil oxygen, foster increased growth of disease causing fungi, and inhibit the cycling of nutrients in the ecosystem. Other recorded effects of low sulfur dioxide levels from the Colstrip studies include reduced seed germination and reductions in the number and variety of plant species (EPA, 1978).

The Colstrip results are supported by other experiments with three important pasture grasses grown in Montana, perennial ryegrass, ricegrass, and orchard grass. Crittenden and Read (1978) demonstrated that an annual ambient sulfur dioxide concentration of 0.037 ppm caused a 36 percent loss in the total dry weight of perennial ryegrass. Bell and Clough (1973) observed a 52 percent yield loss (measured as dry weight of shoots) in ryegrass at a sulfur dioxide concentration of 0.067 ppm after 26 weeks continuous exposure for eight hours a day. When 0.12 ppm sulfur dioxide was administered eight hours a day for nine weeks, there was a 46 percent reduction in leaf and shoot production. Increased leaf drop also was noted at these concentrations and durations. Bell and Mudd (1976) repeated the work of Bell and Clough (1973) demonstrating a 50 percent loss in dry weight of shoots after 26 weeks growth at concentrations of 0.073 ppm sulfur dioxide. Ashenden (1978) found that a four week exposure to 0.11 ppm sulfur dioxide can reduce the growth of orchard grass. Reductions of 34 percent in leaf size and 45 percent in total plant dry weight were found. In a previous work, Ashenden and Mansfield (1977) found a 20 percent decrease in leaf area and a 40 percent reduction in the total dry weight of perennial ryegrass after a four week exposure to 0.11 ppm sulfur dioxide. Finally, Ferenbaugh (1978) found that a six week exposure to 0.06 ppm sulfur dioxide caused reductions in respiration, photosynthesis, and the chlorophyll content of ricegrass. Also noted was a slight increase in plant productivity at 0.06 ppm. This was possibly the result of a fertilizing effect from sulfur dioxide. At 0.13 ppm sulfur dioxide, results showed decreased productivity in ricegrass after six weeks of exposure.

Dose-Response Effects on Timberlands

Scheffer and Hedgecock(1955), in a comprehensive study of timberlands south of a smelter at Trail, B.C., reported extensive injury to shrub and tree species. Their study consisted of field observations of damage and artificial exposure of selected plants to sulfur dioxide. Results showed that artificial exposure of ninebark, spirea, western larch, and ponderosa pine to sulfur dioxide concentrations between 0.53 and 0.60 ppm for seven hours caused significant visible injury. This indicates 0.15 ppm sulfur dioxide for 24 hours would be expected to cause similar injury to these plants (O'Gara, 1922).

It is the consensus among plant scientists that the seedling stage in pines is the most susceptible to adverse effects from sulfur dioxide. Katz and McCallum (1952) found that 0.29 ppm sulfur dioxide caused visible injury to Douglas fir seedlings after 44 hours of exposure. Constantinidou et al. (1976) found that 4.0, 3.0, 1.0, 0.5 ppm sulfur dioxide administered for 15, 30, 60, and 120 minutes respectively, caused reductions in the chlorophyll content of embryotic leaves (cotyledons) of red pine. These effects were found to be reflected in reduced weight of the primary needles of the seedlings. All concentrations caused chlorophyll and weight loss with increasing exposures magnifying the effects. Constantinidou et al. said:

> The data suggest that continuous fumigation with SO_2 at much lower dosages than used in these experiments may have important inhibitory effects on seedling development and on regeneration of plant communities.

Western forest trees considered highly sensitive to sulfur dioxide include Douglas fir, western larch, grand and subalpine fir (Scheffer and Hedgecock, 1955). Western woody shrubs considered sensitive include ninebark, serviceberry, spirea, and mockorange. Other researchers (Katz and McCallum, 1952; Linzon, 1972) have ranked ponderosa pine, Douglas fir, and western larch most susceptible to sulfur dioxide.

Tree species considered to be as sensitive to sulfur dioxide as western trees are white birch, eastern white pine and jack pine (Linzon, 1972). Several studies have related concentration and exposure durations to injury in these trees. Houston (1974) found that a 0.025 ppm sulfur dioxide exposure for six hours caused initiation of needle damage in eastern white pine. Tolerant varieties of the same tree were affected to the same degree by 0.05 ppm after an exposure of six hours. Sensitive varieties of eastern white pine showed tip injury from 0.05 ppm sulfur dioxide after a two hour exposure. At levels of 0.25 ppm sulfur dioxide for two hours, both jack pine and eastern white pine showed damaged needles (Berry, 1971).

In northern Ontario, Linzon et al. (1978) observed elevated sulfur levels in white birch leaves and trace to light foliar sulfur dioxide injury at an average seasonal sulfur dioxide concentration of 0.01 ppm (measured as sulfation rate equivalent). At a growing season average concentration of 0.03 ppm, moderate to severe injury and twice the sulfur content in leaves was observed. Severe injury symptoms and leaf sulfur levels (three times normal) was found associated with an annual sulfur dioxide level of 0.05 ppm.

In a separate set of observations, Linzon et al.(1978) noted that jack pine suffered loss of leaf chlorophyll and tissue death from an annual average sulfur dioxide concentration of 0.045 ppm near copper and nickel smelters at Sudbury, Ontario. A ten year average sulfur dioxide concentration of 0.03 ppm was found to cause an annual saleable timber net loss of 175,104 cubic feet, in eastern white pine near the smelters (Linzon, 1971). Materna (1973) also observed losses in wood growth of fir trees in Germany after the forests were exposed to sulfur dioxide concentrations ranging from 0.006 to 0.009 ppm annual average.

A comprehensive study conducted on the east coast near large fossil fuel power plants indicates that sulfur dioxide in combination with other pollutants can cause excessive damage to plantation grown conifers at low concentrations. An EPA study (1971) found that four month average concentrations of less than 0.01 ppm sulfur dioxide, 0.01 ppm nitrogen dioxide, 0.07 ppb hydrogen fluoride, and 0.05 ppm ozone caused a number of growth abnormalities and visible injury to Scotch pine, Norway spruce, Douglas fir, and Frazier fir.

Dose-Response Effects on "Lower Plants"

Two so called "lower plants" which are particularly sensitive to sulfur dioxide are lichens and bryophytes. Lichens are dual organisms consisting of algae and fungi growing in partnership. Bryophytes represent a class of primitive plants, of which the best known are mosses. Both bryophytes and lichens are important components of Montana plant communities, necessary for the survival of all major agricultural and forest ecosystems in the state. They are pioneer organisms in the formation and maintenance of fertile soil. They help prevent soil erosion, and are used as shelter and food by many types of animals including insects, rodents, birds, elk, deer and moose.

Field observations near Sudbury, Ontario found the significant absence of many lichen species at annual sulfur dioxide concentrations of 0.01 to 0.02 ppm (LeBlanc _et_ _al_., 1972). In this same area a six month average sulfur dioxide level between 0.042 and 0.030 ppm caused some tissue death in lichens. Levels between 0.006 and 0.018 ppm resulted in loss of chlorophyll and growth reductions (LeBlanc and Rao, 1973). A field study in the Tyne valley of England showed that most bryophytes do not survive when the average winter concentration of sulfur dioxide exceeds 0.017 ppm (Gilbert, 1968).

Studies under laboratory conditions have found that sulfur dioxide induced changes in lichens and bryophytes apparently result from alteration in photosynthetic pigments and a decrease in respiration (Puckett, 1973; Braddeley _et_ _al_., 1971).

The absence of lichens and bryophytes in the field also could result from a lack of reproductive capacity. LeBlanc and Rao, (1973) reported that lichens in areas with an annual sulfur dioxide concentration above 0.03 ppm had no reproductive organs. Those growing in areas with 0.005 to 0.03 ppm sulfur dioxide, had few such organs while those growing in areas with less than 0.005 ppm sulfur dioxide had normal reproductive capacity.

Effects of Particulates on Agricultural and Other Plants

The effects of particulates on plants are not understood as closely as those from gaseous pollutants. Studies indicate that the effects of particulates result from their chemical properties or from the interference with the biological functioning of plants (Darley, 1966). If particles are chemically reactive they may damage plant surfaces. If particles are very small, they may enter the stomata and hinder respiration. The effect of fluoride particulates on plants are discussed on pp.204.

Cement Kiln Dust

Harmful effects from cement kiln dust result from formation of a crust on limbs and leaves when settled dust combines with moisture. Czaja (1962, 1966) said this crust results from the hydration of calcium silicates (a constituent of cement-kiln dust) to form a crystalline lattice completely enclosing the leaves and preventing normal function. Darley (1966) observed a reduction of spring growth in conifers where the oldest needles were encrusted. Anderson (1914) demonstrated that dust from a cement plant in New York accumulated on cherry tree stomata, reducing pollen germination and fruit set.

The physical mechanism causing vegetation injury results from reduced photosynthesis and starch formation. Steinhuabel (1962) found that dust-covered holly leaves underwent a reduction in starch content resulting from reduced light absorption. Czaja (1966) presented evidence indicating that stomata in conifers could be clogged by dust, preventing the free exchange of carbon dioxide and oxygen, which is necessary for normal plant growth and development.

Chemical reactivity of cement-kiln dust also causes plant damage. Czaja (1962) found the hydration process associated with crust formation releases highly alkaline calcium hydroxide. He found that the alkaline solution either penetrates through the stomata or dissolves the protective layer of the leaf and injures internal cell structures. Darley (1966) showed that cement-kiln dust at 0.011 gm/cm^2/month caused reductions in carbon dioxide absorption and that 0.014 gm/cm^2/month caused moderate leaf injury. Darley misted water on the plants during dust exposure. Plants not misted did not show injury.

Industrial Soot

Berge (1965) found that soot deposition on conifers near Cologne, Germany plugged stomata and caused growth losses. Dead leaf tissue was observed on the leaves of several plant species in a greenhouse where soot from a nearby smokestack entered (Miller and Rich, 1967). The spotting resulted from the acidity of the soot particles. Plants outside the greenhouse were not damaged, possibly because rain washed off the particles before they could cause damage.

Coal Dust

Auclair (1976) determined that coal dust can be toxic to vegetation. Working with spruce, he found that deposition of coal dust on the needles reduced photosynthesis and in time could kill the plant. Rao (1970) found that dustfall levels of 1.90 gm/cm^2/month from coal unloading caused 25 to

55 percent damage to mango tree leaves, and 10 to 15 percent damage to lemon tree leaves. Dead leaf buds and loss of leaves also were significantly increased for both trees. Rao also reported that the fruit production from both mango and lemon was reduced to levels well below prepollution periods.

Sulfates

Sulfates formed from sulfur dioxide in the atmosphere eventually settle out of the air. Sulfate particles less than one micrometer behave like gases. Plants can absorb these small particulates through leaf stomata or from deposition on leaves and subsequent entry through the leaf surface (Spedding, 1969). A number of researchers have determined that long term injury to vegetation results from excessive sulfate accumulation (Thomas, 1961). Plants already exposed to sulfur dioxide or hydrogen sulfide or in areas with elevated soil sulfate are more inclined to injury from sulfate.

Larger sulfate particles also can injure plants during sensitive periods in their life cycle. Evans et al. (1977, 1978) studied the effect of neutral (pH=7) sulfates on vegetative and sexual development in bracken ferns. Results showed that small amounts of sulfate decreased sperm motility. Additions of sulfate increased the effect. Loss of sperm motility indicates a reduction in the reproduction potential of the fern, probably resulting in lower numbers of these plants in high sulfate areas.

Sulfuric Acid Mists

Sulfate dissolved in water droplets smaller than 400 micrometers in diameter is called acid mist. These small particles are formed near sulfur dioxide sources. Larger water droplets precipitate as acid rain.

Acid mists are known to damage vegetation. Hindawi _et_ _al_. (1977) observed the effect on bush beans of one 45 minute acid mist treatment per week for six weeks at five acidity levels ranging from pH 5.5 to 2.0. The mist had a size range of 4 to 120 micrometers and a settling rate ranging from 0.006 to 0.6 micrograms sulfuric acid per square centimeter per minute. Visible symptoms of injury on plants occurred below pH 3.0. Leaf dry weight appeared to be the most sensitive growth loss indicator. At pH 2.0 there was a 33 percent leaf dry weight reduction. A ten percent reduction occurred at pH 3 and 4 without visible leaf injury. At this acidity level there also was an approximately ten percent loss in total dry weight. Hindawi _et_ _al_. also found the application of acid mist resulted in significant losses in leaf nitrogen, phosphorous, magnesium, and calcium. Potassium appeared unaffected and sulfur accumulation increased on leaves and soil after acid mist treatments.

Wood and Bormann (1974) studied the effect on birch seedlings of acid mists with a diameter of 25 to 100 micrometers and pH of 5.5, 4.0, 3.3, 3.0, 2.3. Exposures lasted for six hours and were repeated once a week over 120 days. Results indicated that two-week old seedlings were more susceptible to aerosol damage than six-week old seedlings. Wood and Bormann suggest that plants with developing tissues are most susceptible to acid mist damage. In another experiment, Wood and Bormann (1975) exposed sugar maple and pinto bean plants to sulfuric acid mists and found that mist with a pH of 3.3 caused increased leaching of leaf calcium, potassium, and magnesium without visible injury.

Lang _et_ _al_. (1978) found in experiments with a number of plants that aerosols with a concentration of 100 micrograms sulfuric acid/m^3 and diameter of 0.3 micrometers entered the plant directly through leaf stomata.

They found damage from these exposures to be most severe with bean and poplar, less serious with soybean, green ash, and birch, and least serious with corn and wheat.

Effects on Livestock and Other Animals

Sulfur Dioxide

There appear to be no data in the literature on the effect of sulfur oxides on livestock and wildlife. Effects similar to those reported for laboratory animals in the Human Health section probably would be found among livestock at similar sulfur dioxide concentrations.

Particulates

It has been shown that animals are affected by eating vegetation contaminated by heavy metal particulates such as lead (NAS, 1972). They also may be affected by other metallic contaminants. Erdman *et al*. (1978) said that molybdenosis (a disease caused by excessive molybdenum consumption) may result when cattle forage on vegetation with excessive levels of this metal.

Like humans, cattle may be susceptible to respiratory impairments when particulates are inhaled and retained in the lung (Lillie, 1970). In a recent Department of Health and Environmental Sciences public hearing, Dr. Herb Smith (a Colorado veterinarian) said there is "consistent increased susceptibility to respiratory infection following particulate matter insult" (DHES, Public Hearing, March 11, 1978).

Insects are especially susceptible to accumulation of trace elements from particulate fallout. Bromenshenk (1978) summarized the literature and found that pollinators (especially honeybees) are the most sensitive insects because of their distribution, behavior, and feeding habits. He found that reductions in forest floor insects, ground beetles, and beetle predators have been reported where metals such as cadmium, copper, lead or zinc have accumulated.

Effects on Ecosystems

Acid Precipitation

Oxides of sulfur and nitrogen emitted into the ambient air react with water vapor to form acids. These acids may fall to earth as acidic rain, snow, or other forms of precipitation. Pure water is neither acidic nor basic, and therefore, has a neutral pH of 7.0. A pH less than 7 indicates a substance is acidic, and a pH above 7 indicates a substance is basic. Rainfall not contaminated by pollution normally is slightly acidic, with the interaction between naturally occurring carbon dioxide in the atmosphere and rainwater resulting in a pH of approximately 5.7. Rainfall with a pH of 5.6 or lower has increased with large scale utilization of fossil fuels in electric power generation and industrial development (Shriner *et al.*, 1977).

Thousands of square miles in the eastern United States commonly experience rainfall averaging pH 4.4 with individual measurements as low as pH 3.0 (Likens, 1976). Similar measurements have been made in Washington state (Denison *et al.*, 1976). Evidence presented at the First International Symposium on Acid Precipitation and the Forest Ecosystem (Dochinger and Seliga, 1976) showed that sulfur dioxide and other pollutants are often transported far downwind from their sources. There is documentation of pollutants being carried 600 miles from industrialized areas in Germany and England to southern Scandinavia, where they precipitate out as acid rain.

Aquatic ecosystems have shown the most adverse effects from acid precipitation. Acidity has increased 100-fold in lakes of southeastern Canada in the last 10 years. Researchers have noted a correlation between decreases in fish populations and increased acidity. In water where the pH ranged between 4.7 and 5.2, most fish species failed to reproduce (Beamish, 1974). Similar studies have shown the same phenomenon in the Scandinavian countries. Thousands of lakes and rivers there have become acidic to the point where they are devoid of salmonid fishes. Acidification of these waters adversely affects bacteria, microorganisms, and aquatic insects, all important components of lake and stream ecosystems (Dochinger and Seliga, 1976).

Vegetation also is directly damaged by acid precipitation. Acid rain can affect the biological function of plants leading to tissue death (Ferenbaugh, 1975), or dissolve the wax coating on flower petals and leaves making them more susceptible to disease and other environmental stress factors (Lang _et_ _al_., 1978). Rain with pH low enough to cause immediate damage to vegetation rarely is found in the United States. However, once the acidic rain has fallen and landed on a plant, evaporation causes increased acidity with an increased potential for damage to vegetation (Gordon, 1972).

Indirect effects of acid precipitation on plants, also have been recorded. Among these are the alteration of nutrient availability in soils (Tamm and Cowling, 1976), changes in species composition of plant communities (Ferenbaugh, 1975), and increased uptake of soil contaminants such as cadmium (Shen-Miller _et_ _al_., 1976).

Evans _et_ _al_. (1978) studied the effects of simulated acid rains on several plants. With populations of hybrid poplars, simulated sulfuric acid rains from pH 2.7 to 3.4 caused vegetation damage. At pH 2.7, 10 percent of the leaf area was injured by a dose of five rains lasting six minutes each on five consecutive days. At pH of 3.4, one percent injury occurred. Testing green beans and sunflowers with an artificial rain mixture similar to that derived from coal-fired power plant emissions, Evans and coworkers (1977) observed damage to leaf surfaces at pH 2.7 and 3.0. Damage to leaf cells adjacent to leaf hairs (trichomes) and stomata were observed after a single exposure to simulated rain of pH 2.7. On sunflower at pH levels of 2.3 to 3.1, developing plants in the 3-4 leaf state were more sensitive than plants with five to eight leaves. When plants were mature enough to flower, there was no injury from simulated acid rain at any level tested.

Conifers appear highly susceptible to acid precipitation injury during certain portions of their growth cycle. Gordon and Tourangeau (1975) studied the effects of applications of simulated rain at pH 4.5 for 20 to 30 days during needle formation on Scotch pine and several types of western pine trees. They found several types of needle deformities similar to those found on trees near large sulfur dioxide sources in the United States.

The effects of acid precipitation on moss and lichens also have been investigated. Sheridan and Rosentreter (1973) studied the effects of simulated acid rain on the chlorophyll content, photosynthesis, and respiration of moss. Results indicated that photosynthesis was reduced proportionally as the pH of the rain water decreased. Respiration and total chlorophyll decreased markedly below a pH of 3.0. Dennison et al. (1975) found that lichens and many other small organisms exposed to pH levels below 6 showed decreased nitrogen fixation. Nitrogen fixation by these organisms is an essential preliminary step for the normal growth of many other components of this ecosystem. Reductions in nitrogen fixation by lichens was expected to reduce the amount of nutrients available to other forms of vegetation.

Several studies have noted that acid precipitation increases the loss of calcium, magnesium, potassium, and other nutrients from plant leaves (Wittaker et al., 1974). Those researchers stress that nutrient loss from vegetation can affect plant growth and nutrient cycling throughout the ecosystem.

The most serious long-term consequences of acid precipitation may result from acidification of the soil. Bolin et al. (1971) and Overrein (1972) reported in separate studies that increasing soil acidity stimulated the loss (leaching) of calcium and other nutrients. It appears that soils in high precipitation areas are most vulnerable to nutrient leaching, but experiments also showed effects on dry land ecosystems.

The degree of soil acidification depends in part on soil characteristics. Soils suited to farming are basic (pH above 7.0) and initially more resistant to increases in acidity. Forest soils, on the other hand, already are acidic under natural conditions. The addition of acid precipitation to these soils rapidly tends to increase their acidity. Oden (1968) said a 30 to 50 year period is sufficient to severely increase the acidity of forest soils. More acidity means reductions in plant nutrients. This in turn means leass productive soils and reduced potential for plant growth.

In Sweden, Johsson and Sunberg (1972) observed significant growth loss in trees growing in highly acidic soils. Bolin et al. (1971) estimated this decrease to be 0.2 percent per year. They said continued acid rainfall will cause tree growth decline to reach 15 percent by the year 2000. Whittaker et al. (1974) found that hardwood forests in New Hampshire are declining due to acid precipitation. They found a 19 percent decrease in wood volume growth since 1960.

Acid rain, more than any other effect of sulfur oxides pollutions has been shown to act cumulatively to change the structure of ecosystems. Plants, soil nutrients, microorganisms, acidity, bacteria, and fungi are all affected, with drastic implications for future productivity and possibly the existence of plant communities. These implications, together with long distance transport of this pollutant from its sources, suggest the possibility of global scale impacts on land and water ecosystems.

Particulates

The deposition of particulates may affect the structure of ecosystems. Brandt and Rhoades (1972) found that a forest community recently exposed to particulates showed a significant change in growth forms and in the number of shrubs, seedlings and saplings. These researchers hypothesized that the long-term impact of particulates would be a shift in the composition and dominant species in the forest. Gilbert (1975) observed similar structural changes in lichen communities impacted by particulate fallout.

Grassland ecosystems also may be adversely affected by particulate levels. Kovar (1977) found that a grassland ecosystem's structure and yield were affected by elevated particulate levels. These particulate levels also resulted in changes in water availability and growing conditions which would tend to favor a shift from grass to nongrass plant species.

Weather Modification

Particulate pollution also is believed to affect local weather patterns. It has been suggested that particulates may affect long-term global climate, but this remains uncertain (Hobbs _et_ _al_., 1974).

Increased clouds and fog may be caused by an increase in particulates. Sulfur dioxide also is involved in one cloud forming mechanism hypothesized by scientists. In this process, sulfur dioxide becomes attached to a particle and converts to sulfuric acid, making the resulting droplet highly attractive to water vapor. This droplet could then provide a center for the growth of large fog droplets or rain.

The best known example of weather modification caused by air pollution is the "LaPorte weather anomaly." This was demonstrated by a sequence of careful studies which found that rainfall fluctuations in LaPorte, Indiana, correlated strongly with changes in steel production and consequent particulate emission in Chicago, Illinois, and Gary, Indiana. Air pollution controls now have reduced the rain fluctuations (Changnon, 1968). The size and concentration of the particles is important in causing increased rainfall. Other investigations have shown particulates causing reduced rainfall or exerting no observable effect (Hindman, 1977).

Changes in the local temperature structure of the atmosphere can be caused by suspended particulates. These changes may stabilize temperature inversions, trapping pollution under them and creating conditions favorable to potentially dangerous air pollution episodes (Ackerman, 1977).

Effects on Materials

When sufficient humidity is present, sulfur dioxide and particulate sulfates convert to sulfuric acid and can cause extensive damage to materials. Other particulates can form a coating on materials, trapping moisture and accelerating acid erosion.

In an extensive analysis of the materials damage done by various air pollutants (Salmon, 1970), sulfur dioxide was found to contribute to the deterioration of metals, cotton, finishes and coatings, building stone, paints, paper and leather. Combinations of particulates and sulfur dioxide damage stone, clay and glass. The most significant effects are on paint, stone, and both galvanized and untreated iron and steel.

Haynie and Upham (1974) extensively researched the corrosion of steel during both long and short term pollution exposures. They established that the important contributors are sulfur dioxide, the sulfate content of particulates, and relative humidity. They determined a relationship between the depth of corrosion and these pollutants. The rate of corrosion was found to increase rapidly as the amount of sulfur dioxide present increased.

The zinc coating of galvanized steel normally corrodes lightly to provide a protective coating. However, sulfur dioxide removes some of the normally protective compounds. The presence of humidity sufficient to wet the surface is again important. The rate of corrosion increases as the sulfur dioxide concentrations increase (Guttman, 1968).

Limestone, sandstone, and some other stone materials are particularly vulnerable to sulfur oxides. Sulfuric acid reacts with the stone to form a compound soluble in water. In some stones the chemical reaction can cause an expansion of the stone which results in cracking and further damage.

Soiling of insulators on high-voltage power lines can cause severe problems. When a conductive coating builds up on the surface a short to ground can occur, possibly damaging the insulator or shorting out an entire circuit (Lambeth, 1971).

Building materials are soiled by deposits of particulates and damaged by attempts to clean them. In an extensive study of damage to building materials, Beloin and Haynie (1975) found that asbestos shingles and painted siding were easily soiled by particulate matter, but that concrete, limestone, and window glass were less susceptible.

Laboratory and field tests have confirmed that oil-based house paints can be severely damaged by sulfur dioxide. Latex paints are less susceptible. Many industrial coatings have bases similar to those of oil-based house paints and are similarly damaged (Campbell et al., 1974).

Soiling of paint by the deposition of soot and other suspended particulates has been shown to increase the need for cleaning and repainting. A survey by Michelson and Tourin (1967) of five communities with different levels of suspended particulate pollution found that paint damage likely was related to particulate levels. Specifically, they observed that maintenance intervals at 60 ug/m^3 TSP were about four years, while at 235 ug/m^3 TSP, they were less than one year.

Public attitudes toward air pollution have been found to be closely related to the amount of particulate matter that settles to the ground. Carefully interviewing the residents of more than 7200 households in Birmingham, Alabama, Stalker and Robison (1967) found 33 percent of those interviewed were annoyed by air pollution in areas where the amount of settled particulate was 10 grams per square meter (gm/m^2) per month. Seventeen percent were annoyed when the fallout was 5 gm/m^2 per month.

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III.B. LEAD

Introduction

Lead is emitted from engines burning lead-additive gasoline and from copper and lead smelters. High ambient concentrations in Montana have been measured near the ASARCO lead smelter in East Helena and the Anaconda copper smelter in Anaconda. Additional information on the sources and ambient measurements of lead in Montana can be found in Section II.B and Appendix A.(Hubbel, 1978).

Lead concentrations as high as those recorded near the ASARCO lead smelter have been observed to adversely affect human health. Lead interferes with the formation of hemoglobin and measurably reduces the ability of children to perform intelligence and coordination tests. The segment of the population most sensitive to lead exposure is children, especially those under the age of three years.

Adverse effects on animals also have been observed from exposures to soil and air lead in Montana. Grazing animals have died from lead poisoning near the ASARCO lead smelter.

Human Health Effects

People take in lead from a variety of sources in the environment. Lead is present in water supplies, canned and fresh food, and as a contaminant in the atmosphere. Children can also take in lead from soils and peeling paint. The quantities actually absorbed by the body are determined by a variety of conditions such as the chemical form of the lead, nutritional status, age, and history of previous exposure.

Clinical studies have shown that as much as 32 percent of inhaled lead will be deposited in the lungs (Natusch and Wallace, 1974). Up to fifty percent of this is absorbed into the blood stream (Chamberlain *et al.*, 1975).

The remaining fifty percent is moved to the throat by the mucous lining of the upper lungs. It is then swallowed into the digestive system. In children, forty percent of the lead taken into the digestive tract is absorbed into the blood, compared to ten percent absorbed in adults (EPA, 1977). Clinical studies by Manton (1977) found that between 7 and 41 percent of the total daily lead intake in adults could be attributed to airborne lead. In a similar study, Rabinowitz _et al_. (1973) found 33 percent of the daily lead intake coming from the air.

Indirect sources of atmospheric lead are especially important with children. Lead deposited from the air on soil and in household dust can be picked up on children's hands during play and taken in through the mouth by thumb sucking or other mouthing behavior. Children with a craving for non-food substances (pica) are especially prone to this form of lead uptake (Landrigan _et al_., 1976). Yankel _et al_. (1977) found household dust and soil to be a major source of lead in the blood of children in the vicinity of an Idaho lead smelter. They considered children to be most susceptible between the ages of 18 months and nine years, with sensitivity decreasing with age.

Red blood cells carry lead to soft tissues (primarily the liver and kidneys) and bone, where it accumulates. Lead concentrations in the skeleton reflect long-term cumulative exposure. Elevated lead levels in the soft tissue and blood reflect more recent exposures. Since lead is more harmful in the soft tissue and blood than in bone, blood lead levels are used as a monitor to indicate harmful levels in the human body.

From an air quality standpoint, the most important health effects of lead are on the central nervous system, kidney, liver and blood. Among lead's effects on blood, one of the most serious is anemia, characterized by low hemoglobin levels and increased destruction of essential components of hemoglobin. Tola et al. (1973) reports that less than 50 micrograms of lead per deciliter of blood (ug/dl) is capable of decreasing the hemoglobin level, initiating anemia in adults. Similarly, Rosen et al. (1974) and Pueschel et al. (1972) noted decreased hemoglobin in children as blood lead levels rose above 40 ug/dl.

The Center for Disease Control (CDC) of the U. S. Public Health Service, has defined 30 ug/dl as the level indicating the onset of lead poisoning in children (CDC, 1978). Lead poisoning inhibits two important steps in the formation of hemoglobin. One of these is the inhibition of the enzyme ALAD. This enzyme helps assemble a molecule which eventually becomes a part of hemoglobin. Lead inactivates ALAD at levels as low as 17 ug/dl (Granick et al., 1973). When blood lead levels are above 55 ug/dl, ALAD is 90 percent inactivated (Hernberg and Nikkanen, 1970). Below 40 ug/dl the effect of lead on ALAD decreases significantly (Selander and Cramer, 1970). Another step in hemoglobin formation inhibited by lead is the inclusion of iron into hemoglobin. Iron is an essential component of the hemoglobin molecule, necessary for oxygen transport. When the inclusion of iron is interrupted by lead, a compound called erthrocyte protoprophyrin (EP) is formed. EP is a direct indicator of a disruption in normal hemoglobin production (Piomelli et al., 1973). Roels et al. (1976) calculated the threshold for elevated EP in children to be near 15 to 20 ug/dl. Production of EP was defined by the CDC as the critical indicator of lead levels sufficient to define lead poisoning in children.

The effects of lead exposure on the central nervous system range from acute lead poisoning and fatal brain damage at high levels to subtle physical and mental changes at lower exposures. Irreversible brain damage usually occurs in adults at blood lead levels greater than 120 ug/dl (Kehoe, 1961), although such damage has been recorded at lower levels (Smith *et al*., 1938). The child's brain, because it is still developing, is more vulnerable than that of an adult. The blood lead level causing the onset of brain damage in children is approximately 100 ug/dl, occasionally ranging as low as 80 ug/dl (Bradley *et al*., 1956; Bradley and Baumgartner, 1958). Perlstein and Attala (1966) found that 82 percent of children surviving lead-caused brain damage suffered irremediable spasms, mental retardation and seizures.

Impaired mental performance has been found to occur at low blood lead levels. Perino and Ernhart (1974) found that preschool children with blood lead levels of 40 to 70 ug/dl performed significantly less well on a standard intelligence test than children with blood lead levels between 10 and 30 ug/dl. Landrigan *et al*. (1975) found that children with blood lead levels above 40 ug/dl performed worse on intelligence tests than children with lower blood lead levels. More recently, IQ testing by Needleman *et al*. (1978) found significantly poorer mental performance in one group of children with blood lead levels averaging 35 ug/dl than in a group with average blood lead levels of 24 ug/dl. There were no significant socio-economic differences between the families of the two groups of children. These studies indicate that children without outward signs of lead poisoning can have impaired brain function resulting in behavioral defects.

Excessive lead exposure can result in short and long-term kidney damage in people. A threshold blood lead level where effects begin is not definable for either short or long-term exposures but the effects usually are reversible with medical treatment and cessation of exposure (Hammond *et al*., 1972).

In summarizing the data on human health effects, it is important to identify the portion of the population most susceptible to health damage from lead pollution. Table III.B-I. summarizes the relationship between blood lead levels, observed effects, and the portion of the population affected. It is clear in this table that children show adverse effects at blood lead levels lower than the general population. The Environmental Protection Agency (1977) cites seven possible reasons for the increased susceptibility of children:

> . . .(1) greater lead intake of infants on a per unit body weight basis, which is probably related to greater caloric and water requirement; (2) greater intake as well as net absorption, greater net respiratory intake as well as greater net absorption and retention from the gastrointestinal tract; (3) rapid growth rate may reduce the margin of safety against a variety of stresses including iron, calcium, and vitamin deficiency; (4) dietary habits of children are quite different from adults; normal hand-to-mouth activity probably results in the transfer of lead-containing dust and dirt via thumb sucking or in retrieval of dirt-contaminated foodstuffs; (5) the metabolic requirements for protein, calcium, and iron are so great relative to intake that a negative balance in these factors may exist; (6) in very young children metabolic pathways and factors such as the blood-brain barrier are known to be incompletely developed; and (7) partitioning of lead in the bones of children is different from that of adults. Only 60 to 65 percent of the lead body burden is in the bones of children, and this fraction may be more labile (susceptible to movement).

Pregnant women and the unborn also have been categorized as more susceptible to lead pollution than the normal populace. The increased risk to the unborn appears related to the ability of lead to transfer across the barrier between the mother and unborn child. In the case of the mother, the risk relates to the physical stress of pregnancy. Data suggests that prenatal exposure is associated with mental retardation and a greater instance of abortions and stillbirths (Moore _et al_., 1977; Wibberley _et al_., 1977). The level necessary to cause these effects is not known.

TABLE IIIB-I. SUMMARY: HEALTH EFFECTS OF LEAD

Population Group	Lowest Lead in Blood for Observed Effect (ug/dl)	Observed Effect	References
Children and adults	less than 16	ALAD inhibition	Hernberg and Nikkanen, 1970
Children	15-20	EP elevation	Roels *et al*., 1976
Children	40	ALA elevation	Selander and Cramer, 1970
Children	40	Initiation of Anemia	Rosen *et al*., 1974 Pueschel *et al*., 1972
Adults	50	Initiation of Anemia	Tola *et al*., 1973
Children	35-40	Decline in mental abilities	Needleman *et al*., 1978 Perino and Ernhart, 1974
Children	80-100	Brain damage symptoms	Bradley *et al*., 1956 Bradley and Baumgartner, 1958
Adults	100-120	Brain damage symptoms	Smith *et al*., 1938 Kehoe, 1961

Blood Lead-Air Lead Relationships

In order to develop an ambient air standard sufficient to protect against the effects of airborne lead, it is necessary to know the relative percentages of total blood lead resulting from airborne lead, food, water and other sources. Information on the avenues of lead absorption from sources other than air is very difficult to obtain. Nonetheless, some generalizations can be made. Data collected by Hammer et al. (1971) indicate that children in Helena, Montana exposed to 0.1 ug/m^3 air lead had blood lead levels of 11.6 ug/dl. Angel (EPA, 1977) found a mean blood lead level of 14.4 ug/dl associated with 0.14 ug/m^3 lead in air in Omaha, Nebraska. Finally, Johnson et al. (1975) found female children in rural California with blood lead levels of 10.2 ug/dl at ambient air lead levels of 0.6 ug/m3. These studies suggest that at low air lead concentrations, the contributions from other sources than air lead are in the range of 10.2 to 14.4 ug/dl.

Increases in blood lead levels caused by airborne lead do not precisely parallel increases in ambient air lead concentrations. Azar et al. (1975) attempted to define this relationship and found that small increases in low air lead levels raised blood lead by a greater percentage than equal increases at higher air lead levels.

Yankel et al. (1977) studied the decrease in blood lead in children living near an Idaho smelter as new air pollution controls and soil treatments reduced air lead concentrations over a two-year period. The children were divided into four groups, according to how near the smelter they lived. Those nearest the smelter experienced a three-fold reduction in blood lead. The two middle groups had about a two-fold reduction, and the groups farthest from the smelter showed an eight-fold reduction.

The Environmental Protection Agency (1977) analyzed these and other studies and concluded:

> In general the data show that the blood lead to air lead ratio is not constant over the range of air exposure encountered (it varies in most studies between 1 and 2), that males appear to have a higher ratio than females, and that children have slightly higher ratio than adults. Also, the data suggest that more attention should be given to studies of decreasing air lead concentrations in that ratios derived from such studies appear higher than those of steady state or increasing exposures.

In Montana the major study associating levels of lead in soil and air to blood lead levels was carried out near the ASARCO smelting complex in East Helena. The study showed that air lead measured in 1969 averaged 3.9 ug/m^3 at East Helena City Hall and 0.1 ug/m^3 for Helena (Huey, 1972).

Lead in the first inch of the soil surface was found to be 4000, 600 and 100 micrograms per gram (ug/g) at respective distances of 1, 2, and 3 miles northeast of the smelter (Miesch and Huffman, 1972). Blood lead levels in 10 year-old children were found to be higher in East Helena than in Helena, averaging 15.6 ± 5.1 and 11.6 ± 4.0 ug/dl, respectively. No adverse health effects were noted, although blood lead levels indicated an elevated exposure (Hammer et al., 1972).

The State Department of Health conducted a study of blood lead levels in East Helena children for the Center for Disease Control (Eden, 1975). Ninety children were tested, ranging in age from 18 months to ten years (average 6.3 years). Blood lead levels varied from 15 to 62 ug/dl. Seven percent of the children had more than 40 ug/dl with one seven-year old child having a level of 62 ug/dl. Twenty-three percent had blood lead levels above 30 ug/dl. The study was conducted during a period of heavy snow cover, when blood lead levels would be lower than those expected during periods when children are exposed more frequently to soil and dustfall.

An additional blood lead study was performed on children from various Montana cities (Jankowski, 1975). Thirty-five Anaconda children were tested with 36 percent having blood lead levels over 30 ug/dl. Children from the Boulder River School in Boulder, and from Lewis and Clark County (excluding East Helena) also were tested for blood lead. Results showed that five percent of the Boulder River School children has blood lead levels greater than 40 ug/dl with eight percent above 30 ug/dl. In Lewis and Clark County six percent of the children had more than 40 ug/dl and 13 percent had more than 30 ug/dl blood lead. No documentation on exposure history, hygenic habits or explanations of excessive lead exposure was presented in this study.

TABLE III.B-II. ESTIMATED PERCENTAGE OF ADULT POPULATION EXCEEDING A SPECIFIC BLOOD-LEAD LEVEL AT GIVEN AMBIENT AIR-LEAD EXPOSURES

Air Lead ug/m^3	Percent exceeding blood lead level of: 20.0 ug/dl	30.0 ug/dl	40.0 ug/dl
0.5	15.2	0.6	0.02
1.0	26.2	1.7	0.1
1.5	34.1	2.9	0.2
2.0	40.2	4.1	0.3
3.0	49.2	6.6	0.5
4.0	55.7	8.9	0.8
6.0	64.5	13.2	1.5
8.0	70.3	16.9	2.2

Source: Azar _et_ _al_. (1975)

It is possible to calculate the percentage of a population that will exceed a given blood lead level at certain air lead levels. Based on an equation developed by Azar _et_ _al_. (1975), Table III.B-II indicates the relationship between air lead and blood lead levels in adults. This table does not consider lead from soils and other sources. In areas with soils contaminated by airborne lead, children will have elevated blood lead levels regardless of the current air lead concentrations. Data and methods provided by Yankel et al. (1977) make it possible to calculate the percentage of five-year old children in a smelter community who may be expected to exceed the 40 ug/dl blood level threshold for the onset of anemia in children. Table III.B-III displays the results of these calculations for three soil lead levels. If soil lead levels are 4000 ug/g and air lead 2.0 ug/m^3, which is the approximate levels found in East Helena, then the table predicts that 10 percent of the five-year old children will have blood lead levels greater than 40 ug/dl.

TABLE III.B-III PERCENTAGE OF CHILDREN EXPECTED TO EXCEED A BLOOD LEAD LEVEL OF 40 ug/100 ml FOR VARIOUS AIR AND SOIL EXPOSURES

Air Lead ug/m^3	Soil Lead (micrograms lead/gm soil)		
	500	1000	5000
1	2	4	8
2	3	6	12
3	5	9	15

Source: Yankel et al. (1977)

Effects on Crops and Other Plants

Plants are exposed to lead through their leaves, stems, bark, or roots. Weather conditions such as precipitation and wind modify the amounts of lead that come into contact with plant surfaces while the form of the plant determines the chemical nature and type of material ultimately retained (Heichel and Hankin, 1976).

In one experiment over 95 percent of lead particulate was washed from plants by simulated rainfall (Carlson et al., 1976). Of the remaining five percent of lead particulate on the leaf surfaces, most was in insoluble or inert forms. Little (1973) indicates that only soluble lead forms are significant to plants, since insoluble and inert forms do not penetrate leaf surfaces.

Tree bark accumulates lead and has been used as an indicator of lead accumulation in ecosystems. Rolfe (1974) has shown a five-fold increase of lead levels in tree bark over a 50-year period in central Illinois.

The root system is the major avenue of absorption for lead in plants. The actual amount of lead absorbed from soil depends on the acidity and other soil properties (Zimdahl, 1976). Absorption has been shown to be a passive process that increases with soil lead concentration to a certain level. Once absorption has occurred in root systems, lead appears to be bound primarily in storage cells and subsequently is incorporated into the cell wall with only small amounts moved to other parts of the plant (Malone, et al., 1974).

Most of the studies dealing with lead effects on plants have been carried out in nutrient cultures. The literature conflicts on the beginning point for adverse effects but points out the importance of such variables as physical and chemical soil characteristics in determining the adverse levels (Zimdahl, 1976).

Field experiments have not shown substantial adverse effects on plants from lead pollution alone. The lack of such findings appears to result either from lead concentrations too low to cause observable effects or chemicals in the soil holding the lead and making it unavailable to plants (Zimdahl, 1976). Taskey (1972) described the relationship between elevated soil levels of lead and other heavy metals and the absence of plants near the Anaconda smelter. The soils near Anaconda have been seriously eroded over the years, possibly leading to chemical alteration of soil lead and making harmful levels of lead available for uptake by plants.

The major concern relating to lead pollution in plants is the ultimate effect on humans. Many experiments near smelters (Gordon, 1972) and highways (Schuck and Locke, 1970) show increased levels of man-generated lead in and/or on consumer crops. If these plants are eaten without sufficient washing, serious health hazards can result.

Effects on Livestock and Other Animals

Lead poisoning of domestic animals is a problem frequently found near smelters, mines, and industrial plants (Schmitt *et al*., 1971). Although such poisoning also is possible near highways, little literature is available on the subject.

The effects of lead on biological processes of animals generally include the nervous and blood forming systems and kidneys (EPA, 1977). Hammond and Aronson (1964) estimated the minimum cumulative dose for lead poisoning in cattle to be six to seven mg/kg body weight per day. For horses, the toxic dosage is estimated to range from 1.7-2.4 mg/kg body weight per day (Hammond *et al*., 1972). Besides having a natural susceptibility, horses are vulnerable to lead poisoning because of their close-cropping feeding habits. Horses sometimes pull up plants and eat roots and soil along with the leaves, thereby taking in lead present in the soil (Aronson, 1972).

A study by the EPA (Lewis, 1972) near the ASARCO smelter at East Helena showed that a postmortem of one horse from the vicinity indicated acute lead poisoning. One lung showed evidence of chronic lead or cadmium exposure. Pneumonia and heart disease further suggested heavy metal exposure. Cattle near East Helena also have been exposed to excessive lead. A number of animals tested in the area have excessive blood lead levels. Cattle deaths in at least two instances have been attributed to acute lead poisoning (Hubble, 1978). Soil lead levels from one of these pastures contained 1175 ug/g in the first one inch (Maughan, 1978).

The exposure of small mammals near highways has been studied (Jeffries and French, 1972; Williamson and Evans, 1972). Both studies show a correlation between total body lead and distance from the source. In most cases, body and soil lead levels also were correlated. No lead poisoning was reported in either study.

Measurements of lead levels in pigeons (Tansy and Roth, 1970) and starlings (Martin, 1972; Martin and Nickerson, 1973) indicate that urban birds have higher lead levels than rural birds. Toxicity associated with waterfowl feeding in areas of lead mine wastes have been reported (Chupp and Dalke, 1964). Waterfowl appear to be twice as sensitive to the biochemical effects of lead as man and other mammals (Finley *et al*., 1976).

Effects on Ecosystems

The effects of lead on ecosystems is implicit in the nature of lead cycling in nature. Getz *et al*. (1977) researched the movement of lead in a watershed ecosystem near and surrounding an urban center in southern Illinois. The findings indicated that 92.5 percent of the lead entering the ecosystem remains within it. Lead was retained primarily in the soils, plants and stream sediments. The major portion of the accumulated lead was in urban areas and near high use rural roads. Jennett *et al*. (1977) made a similar investigation near a mining and smelter area in southeastern Missouri. This study found elevated concentrations of lead in soils, vegetation and forest floor litter with storm runoff in streams being the major transport mechanism out of the study area. Water velocity prevented lead concentration in stream sediments but lake sediments at the stream's mouth increased in lead.

Effects on Materials

Material destruction by airborne lead does not appear to be a problem of any significance. Catalytic converters in automobiles are made inoperable by lead in automobile emissions. This necessitates the use of unleaded gasoline in these cars. There is also some evidence that lead has a harmful effect on some types of electric circuits, but this has not been completely substantiated (Hammond _et_ _al_., 1972).

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III.C. CARBON MONOXIDE

Introduction

Carbon monoxide (CO) is an odorless, colorless, tasteless gas produced primarily by the incomplete combustion of fuels such as wood, oil, and gasoline. High ambient concentrations of carbon monoxide recorded in Montana appear to be related to automobile traffic and occur almost exclusively in busy urban areas. Additional information on the sources and ambient measurements of carbon monoxide in Montana can be found in Section II. B and Appendix A in this document.

Carbon monoxide at the concentrations recorded in Montana cities has been observed to adversely affect human health. Carbon monoxide reduces the ability of the body to supply oxygen to its tissues. The segments of the population most sensitive to carbon monoxide exposure are pregnant women and persons whose ability to transfer oxygen to their tissues is already impaired, such as those with some types of heart disease (atherosclerosis).

The effects of carbon monoxide may increase with altitude. At the relatively high elevations of Montana cities (even Billings, for example, is over 3000 feet) the reduced atmospheric pressure results in a lower level of available oxygen in the blood which may also increase the susceptibility of the body to carbon monoxide (EPA, 1978).

No adverse effects on vegetation, materials, or the environment have been shown to result at the levels commonly observed in Montana. Some research has indicated that carbon monoxide may contribute to the threat to the ozone layer in the upper atmosphere.

Human Health Effects

Oxygen is transported from the lungs through the body by a portion of the red blood cells called hemoglobin (Hb). When carbon monoxide is present it may become attached to a hemoglobin molecule in place of oxygen. Not only does it occupy a place that should have carried an oxygen molecule but the size of the carbon monoxide molecule distorts the hemoglobin and makes it more difficult for other attached oxygen molecules to be released. This deprives the body of its necessary oxygen supplies. A hemoglobin molecule carrying carbon monoxide is called carboxyhemoglobin (COHb).

Many of the effects of carbon monoxide appear to be caused by simple oxygen starvation of the cells. Carbon monoxide dissolved in the blood also directly interferes with vital energy pathways (oxidative phosphorylation) in the cells. Thus adverse effects are observed at lower concentrations of carbon monoxide than would be expected if oxygen starvation were the only effect (Goldbaum *et al.*,1975).

A certain amount of carbon monoxide is naturally produced in the body. About one-half of one percent (½%) of the hemoglobin normally will be carrying such naturally produced carbon monoxide. This may increase to about one and one-half percent (1½%) in females during menstruation (Coburn, 1977). Smokers also will have a higher level of carboxyhemoglobin due to the carbon monoxide present in the tobacco smoke. During smoking, carboxyhemoglobin levels can increase to about 15 percent (Landaw, 1973). There is some evidence that this higher level of carboxyhemoglobin in smokers may be related to their higher incidence of heart disease (Astrup *et al.*, 1970).

If a person is exposed to carbon monoxide over a prolonged period, the amount of carboxyhemoglobin in the blood will gradually increase. A person (at rest) breathing air containing 30 ppm of carbon monoxide will replace about 2 percent of his total hemoglobin with carboxyhemoglobin after one hour of exposure, increasing to about 3 percent after two hours, and to a maximum of about 5 percent after twelve hours (Smith, 1968). Absorption of the carbon monoxide will be faster if the person is physically active during the exposure. The maximum level of carbon monoxide that will be absorbed into the blood is almost directly proportional to the concentration of carbon monoxide in the air.

A complex mathematical equation can be used to estimate the amount of carboxyhemoglobin that will be present in the blood after exposures of varying time to different concentrations of carbon monoxide in the air (Coburn et al., 1965; Peterson and Stewart, 1975). Table III.C-I shows the effect when an adult female at sea level with a natural burden of eight-tenths percent (0.8%) breathes carbon monoxide in the air for one hour and eight hours. The table lists the different concentrations of carbon monoxide necessary to produce specified concentrations of carboxyhemoglobin for two levels of physical activity.

TABLE III.C-I AMBIENT CARBON MONOXIDE LEVELS AND ASSOCIATED CARBOXYHEMOGLOBIN IN PERCENTAGE AFTER 1 HR. AND 8 HR. EXPOSURES

	Ambient Carbon Monoxide			
	Resting		Moderate Exercise	
Percent Carboxyhemoglobin	1 hr.	8 hr.	1 hr.	8 hr.
1.5	22 ppm	9 ppm	14 ppm	9 ppm
2	36	12	22	12
2.5	50	16	30	16

The table demonstrates that a given level of carboxyhemoglobin in the blood can be produced at various carbon monoxide concentrations by changing the time of exposure. The adverse effects on human health are more directly related to the blood carboxyhemoglobin levels than to the ambient carbon monoxide levels. Therefore the studies reported here will be discussed in terms of the carboxyhemoglobin concentrations. An air quality standard must, however, be expressed in terms of pollutant concentrations in the air. This table will be used to determine the correspondence between concentrations of carboxyhemoglobin and various exposures to carbon monoxide.

Animal Studies

Experiments on animals allow direct examination of the tissues damaged by exposures to carbon monoxide. They also permit investigations of the effects of long-term exposures. A few studies have examined the effects on the central nervous system, while many others have looked at the effects on the heart.

Shul'ga (1969) exposed rats and mice to 26 ppm carbon monoxide for 10 weeks. She observed significant deterioration in spinal cord and brain tissue and inconsistent nerve signal transmission velocities. Preziosi (1970) also observed extensive tissue death in the central nervous system of dogs exposed to 50 ppm and 100 ppm carbon monoxide for six weeks. Some of the changes in the central nervous system did not appear until two weeks after the exposure. Autopsy of the dogs also found significant deterioration of the heart muscles, including thinning of the heart walls and enlargement of the chambers.

A series of studies at the University of Copenhagen has demonstrated adverse changes in tissues of the heart arteries following carbon monoxide exposure. Thomsen (1974) found evidence of the beginning stages of heart disease (atherosclerosis) in a group of monkeys that had been exposed for two weeks to enough carbon monoxide to raise their carboxyhemoglobin levels to approximately 21 percent. Similar results were found in rabbits exposed for only four hours with a carboxyhemoglobin level of approximately 9 percent (Thomsenand Kjedlsen, 1974). Extensive damage to other tissues of the heart also have been observed (Kjeldsen et al.,1974). These injuries may contribute to the onset of heart disease.

The course of healing after an induced heart attack was studied in monkeys by DeBias et al.(1973). When exposure to carbon monoxide was kept at levels sufficient to induce 12 percent carboxyhemoglobin in the blood, slower healing of heart tissues was observed, along with abnormal heart beat patterns (elevated P wave and T wave inversion), compared to a control group of monkeys breathing clean air. In a study of short-term exposures (six hours), DeBias et al. (1976) found that monkeys with carboxyhemoglobin levels of about 9 percent were significantly more susceptible to induced irregular heart beat (fibrillation).

Studies of human subjects have demonstrated a clear relation between measured carboxyhemoglobin levels in mothers and lower than normal birth weights of their children (Astrup et al., 1972; Longo and Hill,1977). Studies in animals have demonstrated that the carboxyhemoglobin levels in the blood of the fetus may be as much as 60 percent higher than in the mother (Longo and Hill, 1977). In one early study, rabbits maintained at carbon monoxide levels high enough to yield 18 percent carboxyhemoglobin throughout pregnancy experienced 35 percent still births or early death of newborn. Rabbits with a carboxyhemoglobin levels of 10 percent had a loss of 10 percent and those exposed to clean air had a loss of only 2.5 percent (Astrup et al., 1972). A group of pregnant rats exposed to enough carbon monoxide to raise the maternal carboxyhemoglobin to 15 percent showed central nervous system damage to the young. Significant decreases in movement and activity of the young were observed. Biochemical analysis of brain tissues of the young found markedly lower levels of brain protein and other important substances (Fechter and Annau, 1977).

Clinical Studies

Laboratory studies on human subjects also have illustrated the effects of carbon monoxide on the central nervous system and the heart. Experiments on the central nervous system effects on humans have included studies of eye-hand coordination, time interval estimation, and mathematical and verbal skills.

Halperin _et al._, (1959) demonstrated a distinct loss in ability to distinguish a small red light from a lighted background when the carboxyhemoglobin in the blood exceeded 4.5 percent. The test scores progressively declined as the carboxyhemoglobin level was slowly raised to 20 percent.

Schulte (1963) administered a battery of ten simple mental skills tests and three simple physical skills tests to forty-nine healthy adult males. In seven of the mental skills tests there was a significant correlation between the increase in errors and completion time of the tests and increasing levels of blood carboxyhemoglobin. The increase was clearly apparent above three to five percent carboxyhemoglobin. Schulte concluded that

> alteration of function due to exposure to carbon monoxide occurred earliest in the higher centers of the central nervous system in that area (or areas) of the brain which controls some of the cognitive and psychomotor abilities. This alteration can and does occur at much lower levels of carboxyhemoglobin than those which are necessary to produce subjective symptoms or alter physiological signs. Furthermore, the degree of alteration in psychological abilities may be quite profound before any clinical signs or subjective symptoms are elicited.

Central nervous system effects were measured by Horvath _et al._ (1971) by observing the ability of experimental subjects to discriminate between bright and dim light signals. Significant increases in error rate were noted as the carboxyhemoglobin levels increased from about two percent to about seven percent. Horvath _et al_. concluded

> Persons who sustain increases in COHb saturation of more than about 2% or 3% may become less effective in coping effectively with unexpected events and more liable to perform routine tasks in an inefficient manner. The danger this could pose to drivers or operators of heavy machinery need not be elaborated.

The heart muscle is extremely sensitive to low blood oxygen levels caused by increased carboxyhemoglobin. During exercise all muscles, including the heart, increase their demand for oxygen. This need can be met by increasing either the rate of blood flow or the amount of oxygen the muscle tissues take out of the blood. Unlike other tissues which may extract only 25 percent of the blood oxygen, the heart normally extracts almost all that is available. Thus the heart, which must work harder during exercise, can increase its oxygen supply only by increasing the rate of blood flow through the heart muscles. If the blood vessels in the heart are inflexible or narrowed by heart disease (atherosclerosis), the rate of blood flow cannot increase sufficiently, leading to a heart attack as heart muscle tissues die from oxygen starvation. If the oxygen carrying ability of the blood is reduced by carboxyhemoglobin the death of heart tissue can occur more easily.

The increased removal of oxygen from the blood builds up an "oxygen debt" requiring replenishment of blood oxygen to bring levels back to normal. When the oxygen debt is large cells can compensate by taking their necessary energy supplies from other reserves, which results in an increase of certain biochemical compounds (lactate and pyruvate) in the blood.

Tests of exercise capacity while breathing carbon monoxide have demonstrated a serious decline in the body's ability to do work. Chevalier _et al_.

(1966) found a 14 percent greater oxygen debt after five minutes of work for subjects who had a carboxyhemoglobin level of about 4 percent, compared to performance by the same subjects breathing clean air. Ekblom and Huot (1972) observed a steady decline in the maximum work time on a treadmill, from a 25 percent decrease at about 4 percent carboxyhemoglobin to a 50 percent decrease at about 20 percent. Dahms _et al_. (1975) and Aronow and Cassidy (1975) have obtained similar results.

Individuals whose ability to respond to lowered oxygen levels when the blood stream is already impaired may be seriously affected by exposures to carbon monoxide. Ayres _et al_. (1969) measured the carboxyhemoglobin levels, the oxygen present in the blood, and the lactate and pyruvate production for a group of subjects breathing carbon monoxide while at rest. Of the eleven subjects, four had coronary heart disease. All were brought to a carboxyhemoglobin level of about 9 percent. In all the subjects, the oxygen in the bloodstream decreased by about 20 percent. The healthy subjects were able to respond to the exposure by significantly increasing their rate of blood flow. The coronary patients were able to increase their flow only slightly. Substantial increases of lactate and pyruvate in the blood of these individuals showed that the reduced oxygen capacity of their blood was forcing their tissues to obtain energy from other sources.

Some coronary heart disease patients experience pain in the chest (angina pectoris) during exercise. Two groups of medical researchers tested subjects with stable angina conditions to determine the effect of carbon monoxide in decreasing the time before the onset of incapacitating pain during exercise. Both Aronow and Isbell (1973) and Anderson _et al_. (1973) observed a 16 percent decrease in the maximum exercise time when carboxyhemoglobin levels were raised to about 3 percent. Adverse effects on the heart (S-T segment depression) also were noted. Anderson et al. also observed that at about 4.5 percent carboxyhemoglobin the pain lasted 30 percent longer.

Similar results were found in an experiment on the exercise tolerance of patients with an intermittently painful disease of the arteries of the leg (Aronow et al., 1974) and for lung (chronic obstructive pulmonary) disease patients (Aronow et al., 1977).

Epidemiological Studies

The adverse effects of carbon monoxide exposure have been very difficult to evaluate in general populations. This is partly because high levels of carbon monoxide are found only near heavily traveled streets. In only a few cities (e.g., Los Angeles, Denver and Chicago) are high concentrations of carbon monoxide sufficiently wide-spread that large numbers of people are affected.

Surveys of blood carboxyhemoglobin levels in general populations have been conducted by Stewart et al. (1974), Kahn et al. (1974), and Smith et al. (1975). In Denver, 76 percent of the nonsmokers tested had more than 1.5 percent carboxyhemoglobin. Ten percent had more than 3.5 percent. In Calgary, Alberta the average end-of-workday level for non-smoking office and retail store employees was 1.7 percent, with about 15 percent having levels above 2.5 percent.

One study of 1,000 industrial workers in Finland found a significant correlation between the potential for exposure to carbon monoxide in the workplace and incidence of chest pains that could be diagnosed as angina pectoris. By the nature of their occupations, those exposed to high carbon monoxide levels were also exposed to higher workplace temperatures, which may have been a factor in the increased incidence of angina pectoris (Hernberg et al., 1976).

Most attempts to correlate death rates in the general population with carbon monoxide levels measured at central city monitors have not succeeded. However, Mendelsohn and Orcutt (1978), who included an analysis by specific cause of death, were able to define a statistically significant correlation between carbon monoxide levels and deaths due to heart failure.

Two studies conducted in Los Angeles found a relationship between daily levels of carbon monoxide and death rates. Cohen et al. (1969) compared the recovery of heart attack patients in hospitals in different parts of Los Angeles. Because high carbon monoxide levels are known to be associated with slower healing of heart tissues in monkeys, it would be expected that higher death rates during recovery might be observed when and where carbon monoxide levels are higher. Indeed, Cohen et al. found a statistically significant higher death rate in the hospitals in more polluted areas when carbon monoxide levels were above 7.7 ppm. Hexter and Goldsmith (1971) studied all deaths in the Los Angeles area for four years, 1962-1965. After correcting for temperature and annual cycles, daily levels of carbon monoxide were found to correlate significantly with daily death rates.

A recent study of persons presenting complaints of chest pains at the emergency room of a large Denver hospital found that there were significantly more such complaints on "high carbon monoxide" days as compared to "low carbon monoxide" days. On the high days, the average 24-hour carbon monoxide concentration was 9.3 ppm and on the low days it was 5.9 ppm. Other pollutants were analyzed and found not to be correlated to the carbon monoxide levels or the rate of complaints (Kurt et al., 1978). Table III.C-II summarizes the health effects of carbon monoxide studies.

TABLE III.C-II SUMMARY: HEALTH EFFECTS OF CARBON MONOXIDE

Subjects	Percent Carboxyhemoglobin	Observed Effect	Reference
Monkeys	12	Slower recovery from heart attack	DeBias et al., 1973
Rabbits	10	Litters of CO-exposed mothers showed higher rates of still birth and early death	Astrup et al., 1972
Rabbits	9	Damage to heart tissues observed	Thomsen and Kjeldsen, 1974
Healthy adult males	4.5	Decreased ability to see dim light	Halperin et al., 1959
Healthy adult males	3-5	Decreased ability to perform mental skills tests	Schulte, 1963
Healthy adult males	4	Decreased ability to do work	Chevalier et al., 1966 Ekblom and Huot, 1972 Aronow and Isbell, 1973
Respiratory disease patients	4	Decreased exercise capacity	Aronow et al., 1974
Atherosclerosis patients	3	Decreased exercise capacity	Aronow et al., 1973
Angina pectoris patients	2.5-3	Decreased exercise capacity	Aronow and Isbell, 1973 Anderson et al., 1973
General Population		Increased chest pain complaints in hospital emergency room	Kurt et al., 1978

Effects on Crops and Other Plants

Carbon monoxide effects on plants have been observed only at concentrations substantially greater than those known to be harmful to human health. Because of the limited adverse effects on plants, they will not be discussed further.

Effects on Livestock and Other Animals

Impacts on livestock and other animals from exposure to ambient carbon monoxide can be expected to parallel human health effects. Although many investigations have addressed the effects of carbon monoxide on experimental animals such as dogs and rats in laboratory tests, field investigations on domestic or wild animals have not been reported.

Lacking other information, it may be assumed that concentrations that do not endanger human health would not produce adverse impacts on livestock or wildlife.

Effects on Ecosystems

Carbon monoxide is produced in nature by an atmospheric chemical reaction between naturally-occurring hydroxyl (OH) and methane, or other hydrocarbons that come from plants. The hydroxyl molecules are believed to originate in upper atmosphere chemical reactions and are brought to lower altitudes by movements of air in the atmosphere. Measurements of the background level of carbon monoxide have indicated that the total amount in the atmosphere may be gradually increasing. In the southern hemisphere the background level is about .06 ppm, and about .16 ppm in the northern hemisphere where more is produced by human activities (Seiler, 1974). On a world-wide average about 50 to 75 percent of the carbon monoxide in the atmosphere is due to the combustion of fuels (Seiler and Zankel, 1976). In urbanized areas of the United States practically 100% of the carbon monoxide present results from human activity.

Carbon monoxide is removed from the air by natural chemical reactions. The principal removal reaction is between carbon monoxide and hydroxyl in the atmosphere. Certain micro-organisms in the soil are capable of taking up carbon monoxide, but this action probably accounts for no more than 25% of the total removal of atmospheric carbon monoxide (Seiler, 1974).

Hydroxyl also plays an important role in removing methyl chloroform from the atmosphere. Higher levels of carbon monoxide due to human activity will reduce the amount of hydroxyl, leaving more methyl chloroform in the atmosphere. An increased amount of methyl chloroform may rise to the upper atmosphere, where it may adversely affect the protective ozone layer (Fishman and Crutzen, 1978).

The reaction of carbon monoxide and hydroxyl yields carbon dioxide and ozone. It has been suggested that this reaction may be one source of ozone pollution in the lower atmosphere (Fishman and Crutzen, 1977). Carbon dioxide plays a critical role in establishing the temperature of the earth. The amount of carbon dioxide in the atmosphere is gradually increasing because of fossil fuel combustion and the removal of the forests (Stuiver, 1978). If the amount of carbon dioxide in the air were to double, the average temperature in the polar areas could increase by about 15°F (Manabe and Wetherald, 1975), increasing the melt rate of the polar ice cap, raising ocean levels and leading to potentially catastrophic world-wide consequences.

Effects on Materials

No effects on materials have been recorded.

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III.D. NITROGEN DIOXIDE

Introduction

Nitrogen dioxide (NO_2) and nitric oxide (NO) are present in the atmosphere from fuel combustion, particularly in diesel engines and fossil-fueled power plants. Most nitric oxide is converted to nitrogen dioxide in the atmosphere. Nitrogen dioxide and other nitrogen oxides have a major role in the photochemical reactions that produce ozone.

Nitrogen dioxide is a yellow-brown gas with a sweet, pungent odor. High ambient concentrations of nitrogen dioxide rarely have been recorded in Montana. Higher levels may occur in the future if there is an increase in the number of diesel-powered automobiles and coal-fired power plants. Additional information on the sources and ambient measurements of nitrogen dioxide can be found in Sections II.B and Appendix A.

Nitrogen dioxide at concentrations occasionally recorded in Montana has been shown to adversely affect human health. Nitrogen dioxide exposures can increase the susceptibility to lung infections. Persons most sensitive to nitrogen dioxide are those with asthma or lung ailments (chronic obstructive pulmonary disease).

Nitrogen dioxide has been found to harm crops of the types grown in Montana. Damage to materials also has been recorded. Nitrogen oxides contribute to increased rainfall acidity observed in Europe and the eastern U.S.

Human Health Effects

Nitrogen dioxide is relatively insoluble in water so it reaches the lung without being removed in the moist air of the nose and throat. Low levels of nitrogen dioxide may injure the body's antibacterial mechanisms increasing susceptibility to lung infections.

Animal Studies

Several experiments show that continuous or intermittent exposures of laboratory animals to nitrogen dioxide at levels of 0.5 ppm and above will increase death rates among the test animals exposed to bacteria causing various diseases (such as pneumonia), compared to animals exposed to the bacteria but not to the nitrogen dioxide (Coffin *et al*., 1976; Ehrlich and Henry, 1968).

One explanation for this effect is suggested by the work of Vosin *et al*. (1977) who found a reduction in the activity of antibacterial cells from the lungs (macrophages) exposed to nitrogen dioxide concentrations of 0.1 ppm for 30 minutes. These experiments were performed in cells grown outside the body rather than inside a living lung, so the effects found may not be the same that would occur under normal conditions. The effects are, however, consistent with those observed in animals at higher nitrogen dioxide concentrations.

Lung cell damage caused by nitrogen dioxide has been observed in experiments with animals. Blair *et al*. (1969) reported that concentrations of 0.15 ppm for six to 24 hours per day over three weeks produced significant structural damage to the lungs of experimental mice. In guinea pigs, short-term exposure of 0.5 ppm increased protein concentrations in the urine, indicating a kidney disorder (Sherwin and Layfield, 1974).

Clinical Studies

The perception threshold of nitrogen dioxide odor is approximately 0.11 ppm. Odor is noticeable immediately upon exposure, but then fades as nitrogen dioxide deadens the sensory cells of the nose. Gradual increases up to 27 ppm are necessary before the impairment is overcome and the odor is again detected (Feldman, 1974).

Effects on the central nervous system also are noted at low levels. Concentrations of 0.07 ppm to 0.26 ppm have been found to significantly slow the adaptation of the eye to darkness (Shalamberidze, 1967; Bondoreva, 1963).

Adverse changes in lung function have been recorded consistently at nitrogen dioxide levels above 1.0 ppm for various time intervals in healthy adult subjects (von Nieding *et al.*, 1973). Persons with asthma or chronic lung disease are more sensitive to nitrogen dioxide than healthy individuals. Studies show increases in airway resistance after a 15 minute exposure to 1.5 ppm nitrogen dioxide in persons with lung disease (chronic bronchitis) (von Nieding *et al.*, 1971). Nitrogen dioxide has also been shown to increase the severity of asthma attacks. Orehek *et al*. (1976) found that a 0.11 ppm nitrogen dioxide exposure caused a statistically significant increase in airway resistance in persons with asthma. When these same individuals were given a drug which can induce an asthma attack (bronchoconstrictor), an even greater increase in airway resistance was observed with the 0.11 ppm nitrogen dioxide exposure. Although it is not possible to be sure of the significance of the Orehek observations in relation to spontaneously occurring illness in normal populations, it seems fair to conclude that nitrogen dioxide at concentrations as low as 0.11 ppm with an exposure period of an hour may contribute to adverse reactions in sensitive individuals.

Hackney et al. (1975) found that an exposure to 0.3 ppm nitrogen dioxide in combination with 0.5 ppm ozone for two hours did not decrease lung function more than when ozone was administered alone. For a two hour exposure, with 0.05 ppm nitrogen dioxide, 0.025 ppm ozone and 0.1 ppm sulfur dioxide, von Nieding et al. (1977) also found no significant influence on lung function. However, when a bronchoconstrictor (2 percent acetylcholine) was administered following the exposure, a significant increase in airway resistance was observed.

Epidemiological Studies

A study near Chatanooga, Tennessee, evaluated the effects of high nitrogen dioxide levels caused by emissions from a TNT plant. Evidence indicated that children experienced breathing difficulty and an increase in respiratory disease at an average nitrogen dioxide exposure level above 0.08 ppm (Shy et al., 1970a, 1970b). Increases in lung disease (bronchitis) also were noted in elementary school children and infants in areas with elevated nitrogen dioxide concentrations (Pearlman, 1971).

The Chattanooga studies have been criticized because of unreliable ambient air measurements for nitrogen dioxide and the presence of other pollutants in the study area. The concentrations noted above and in Table III.D-I for the Shy and Pearlman studies are not those shown in the original reports. Instead, the data are from nitrogen dioxide monitoring stations in the vicinity of the Chattanooga study area, where valid measuring techniques were used (EPA, 1976). Although many pollutants other than nitrogen dioxide were present in the Chattanooga area, variations in sulfate and particulate levels among neighborhoods were considerably smaller than corresponding differences in nitrogen dioxide levels. As a result, elevated nitrogen dioxide levels are more clearly associated with health effects than are the other measured pollutants. However, slight differences between nitrogen dioxide and nitrate levels among the neighborhoods in the study area preclude identification of nitrogen dioxide as the sole cause of the observed health effects (Chapman, 1978).

Gas ranges are a known source of household nitrogen dioxide. Melia et al. (1977) found a significantly greater amount of cough, colds going to the chest, and bronchitis in English children from homes with gas ranges than among children from homes with electric stoves. Although no nitrogen dioxide measurements were made it was concluded that nitrogen dioxide caused the increased respiratory illnesses noted in the study. In a subsequent study Melia et al. (1978) compared nitrogen dioxide levels between English homes with gas stoves and others with electric stoves. They found gas kitchens had average nitrogen dioxide levels of 0.07 ppm, compared to 0.01 ppm in electrical kitchens. Wade et al. (1975) found similar concentrations in American homes. Palmes (1977) found a nitrogen dioxide concentration of 0.05 ppm in U.S. homes with gas ranges while electric kitchens recorded levels of 0.01 ppm. If the levels found in these three studies can be related to the adverse health effects noted by Melia et al. (1977) then a nitrogen dioxide hourly average of 0.07 ppm is suggested as a cause of increased respiratory illnesses. Keller et al. (1975), using a smaller population sample than Melia et al. (1977), and without measuring nitrogen dioxide concentrations, could not detect differences in respiratory illness rates between persons from gas kitchen homes and others from electric kitchen homes.

In studies of policemen occupationally exposed to automobile exhaust, Speizer et al. (1973a, 1973b) found nitrogen dioxide exposures with an annual average of 0.06 ppm associated with slight increased respiratory disease in subjects with many years of exposure. The higher nitrogen dioxide levels also were associated with elevated levels of sulfur dioxide, carbon monoxide

TABLE III.D-I SUMMARY: HEALTH EFFECTS OF NITROGEN DIOXIDE

Subjects	Concentration (ppm)	Duration of Exposure	Effects	References
Healthy adults	0.11		Odor perception threshold	Feldman, 1974
Healthy adults	0.07-0.26	Unknown	Significant impairment of dark adaptation in the eye	Shalamberidze, 1967 Bondoreva, 1963
Chronic bronchitis patients	1.5	15 minutes	Increase in airway resistance	von Nieding *et al*., 1971
Asthma patients	0.11	1 hour	Increased airway resistance. Greater increase when NO_2 combined with a bronchoconstrictor.	Orehek *et al*., 1976
7 & 8 yr. old children	0.20	1 hr. peak	39% higher acute respiratory disease than low nitrogen dioxide level community	Shy *et al*., 1970
7 & 8 yr. old children	0.08-0.15*	Annual	Slightly lung function decreases in high nitrogen dioxide area	Shy *et al*., 1970
7 & 8 hr. old children	0.08-0.15*	Annual	Higher incidence of lower respiratory disease in high nitrogen dioxide area	Pearlman *et al*., 1971
School age children	0.07**	1 hour average	Increased coughing, colds going to chest, and bronchitis	Melia *et al*., 1977

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*Concentration measurements from EPA (1976)

**Concentration measurements from Melia *et al*.(1978).

and lead. As a result, the respiratory symptoms could not specifically implicate nitrogen dioxide as a cause.

Giguz (1968) found that trainees employed in fertilizer or chemical manufacturing showed increased respiratory illness and blood abnormalities in their second year of occupational exposure to 0.05 ppm nitrogen dioxide. Studies in the Soviet Union and Czechoslovakia have indicated increased respiratory disease in individuals living near nitrogen dioxide sources. These studies indicate harmful effects occur when average nitrogen dioxide concentrations of less than 0.10 ppm are associated with other air pollutants. One study indicated average levels between 0.02 and 0.07 ppm caused enlargement of lymph nodes and tonsils and changes in blood characteristics (Petr and Schmidt, 1976; Polyak, 1968). These studies have not been reproduced and the results have not been confirmed by other investigators.

Effects on Crops and Other Plants

Vegetation damage from nitrogen oxide exposures is found near industrial pollution sources. Experimenters have studied the effects of industry-generated nitrogen oxide by exposing plants to equivalent concentrations. Cotton, pinto beans and endive plants, experimentally exposed to 1.0 ppm nitrogen dioxide for 48 hours, showed slight leaf damage. Concentrations of 3.5 ppm for 21 hours killed pinto beans and endive and moderately damaged cotton plants (Heck, 1964).

At 0.3 ppm nitrogen dioxide exposures for 10 to 19 days, pinto bean plants declined in growth and increased in chlorophyll content (Taylor and Eaton, 1966). Similar studies with tomatoes at nitrogen dioxide concentrations of 0.15 and 0.26 ppm for 10 and 22 days showed decreases in dry weight and leaf size. Spierings (1971) found that exposure to 0.25 ppm for an entire growing season reduced tomato yields 22 percent. Species of citrus fruits, tobacco and soybean also have been damaged when experimentally exposed to nitrogen dioxide. At nitrogen dioxide concentrations of 0.25 ppm for an eight month period, leaf drop and loss of color in the leaves was observed in navel orange trees (Thompson, 1970). More recent evidence indicates that exposure to 0.025 ppm or less nitrogen dioxide continuously for eight months can cause increased leaf drop and reduced yields of navel orange trees (Thompson _et al_., 1974).

Experimental exposure of agricultural plants has shown that some are more sensitive than others to nitrogen dioxide. The injury threshold for various plants, defined for a given area, is shown in Table III.D-II(EPA, 1971). Increasing the duration of exposure decreases the threshold concentration necessary to cause injury. In general, plants require 20 percent less nitrogen dioxide for every doubling in exposure duration.

TABLE III.D-II SUSCEPTIBILITY THRESHOLD FOR
ACUTE PLANT INJURY FROM NO_2 (EPA, 1971)

Species	Exposure Time (hr)	Threshold Concentration (ppm)
oats	1	4.0
tomato	1	8.0
lettuce	1.5	4.0
pepper	1.5	5.5
alfalfa	4	30.0
bean	4	10.0
mustard	4	20.0
wheat	7	1.0
navel oranges	35	1.0
endive	48	1.0

The combined effects of nitrogen dioxide and sulfur dioxide on plants has been investigated by several experimenters. Tingey _et al_. (1971) found that 0.05 to 0.25 ppm of nitrogen dioxide and sulfur dioxide administered together caused a synergistic leaf injury in plants. Fugiwara (1973) exposed green peas first to 0.1 ppm sulfur dioxide and then to 0.2 ppm nitrogen dioxide and observed effects equal to the sum of individual effects for the two pollutants, but a combination of 0.1 ppm of both pollutants caused synergistic effects. Hill et al. (1974) exposed 87 native cold desert species in Arizona to a mixture of gases containing slightly more than three times as much sulfur dioxide as nitrogen dioxide. The authors observed no synergistic effect with this combination.

Kress and Skelly (1977) used a number of pollutant combinations to determine growth reduction and injury levels for varieties of sycamore and loblolly pine. Varieties selected fell in two categories, either sensitive or tolerant to pollution. Concentrations used in these experiments were 0.01 ppm nitrogen dioxide, 0.05 ppm ozone, and 0.14 ppm sulfur dioxide for six hours daily for 28 consecutive days. Results showed that combinations of ozone, sulfur dioxide, and nitrogen dioxide caused a 15 percent reduction in the growth of the sensitive variety of loblolly pine and a five percent reduction in the tolerant line. A 45 percent growth reduction was noted in the sensitive sycamores, a 35 percent reduction in the more tolerant variety. The extent of growth reduction paralleled visible injury severity in both sycamores and loblolly pines. Exposure to combined ozone and nitrogen dioxide visibly injured loblolly pine but not sycamores.

Effects on Livestock and Other Animals

No data in the literature deals directly with the effect of nitrogen dioxide on livestock and wildlife. Effects similar to those reported for laboratory animals in the Human Health Section probably would pertain at similar nitrogen dioxide levels.

Effects on Ecosystems

A recent study reports that nitrogen dioxide emissions levels play a role in rainfall acidification over large areas of Europe (Vermeulen, 1978). Refer to pp. 97 for a discussion of the effects of acid rainfall on ecosystems.

Effects on Materials

Nitrogen oxides have been known for some time to cause dye fading. A concentration of 0.6 ppm nitrogen dioxide and nitric oxide, in natural gas home clothes dryers has been shown to fade dyes in certain fabrics (EPA, 1971).

Corrosion in nickel-brass alloys has been observed on telephone relay switches in California cities. The corrosion results from nitrate particulate deposits. This type of nitrate particulate accumulation has been associated with an average nitrogen dioxide concentration as low as 0.07 ppm (EPA, 1971).

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III.E. PHOTOCHEMICAL POLLUTANTS

Introduction

Photochemical reactions in the atmosphere produce a characteristic group of pollutants. The most significant is ozone (O_3), a colorless gas with a sharp odor. The other important products of photochemical reactions are hydrogen peroxide (H_2O_2), aldehydes (especially formaldehyde), acrolein, formic acid, and peroxyacetylnitrate (PAN). Ozone and many of these other gases are referred to collectively as oxidants. Fine particulates are also produced by photochemical reactions. As discussed in Section II.A, these reactions can occur whenever enough nitrogen oxides and hydrocarbons are present in the air with sufficient sunlight. The principal sources of hydrocarbons in Montana are motor vehicles and industrial sources such as refineries. Nitrogen oxides come from motor vehicles (especially diesel engines) and fossil-fueled power plants. High ambient levels of ozone have been measured in Billings. Additional information on the sources and ambient measurements of ozone can be found in Section II.B and Appendix A.

Ozone at concentrations occasionally recorded in Montana has been shown to adversely affect human health. Ozone in the air irritates the throat and lungs and impairs breathing. The people most sensitive to ozone are those with asthma or lung ailments and persons engaged in moderate to heavy physical activity.

Adverse ozone effects have been observed on crop types grown in Montana. The effects are increased when plants are exposed to ozone in combination with other pollutants.

Human Health Effects

Animal Studies

Lung damage is observed in experimental animals after long-term exposure to ozone. P'an et al. (1972) found rabbits exposed to 0.4 ppm ozone for six hours a day, five days a week, for 10 months showed substantial lung tissue destruction and decreased oxygen exchange capacity. At 0.54 ppm continuous ozone exposure over these months, Stephans et al. (1976) found lung tissue destruction and an increased stress on the bacterial defense systems of the lungs of laboratory rats. These studies suggest similar effects are possible in humans from long-term ozone exposure.

There is substantial evidence from animal studies that suggests increased susceptibility to bacterial infection following ozone exposure. These studies indicate 0.08 ppm (Coffin et al., 1968) and 0.1 ppm (Miller et al., 1978) ozone is capable of causing increased death rates in mice exposed to a bacterial aerosol. Other experiments exposing mice to various combinations of pollutants such as 0.05 ppm ozone and two ppm nitrogen dioxide for three hours (Ehrlich et al., 1976), or 0.1 ppm ozone, and 0.9 milligrams (mg) sulfuric acid/m^3 for two hours (Gardner et al., 1977) also showed an increased death rate in mice after bacterial infection. Work by Coffin and Blommer (1970) offers an explanation for this effect. They measured the growth rate of bacterial colonies in the lungs of mice following ozone exposure and found that there was significant damage to the infection mechanisms of the lung.

Lung function also has been disrupted by ozone in experimental animals. At 0.02 ppm ozone for 30 days, Bartlett et al. (1974) noted an average lung volume increase of 16 percent and a decrease in lung tissue elasticity in rats. Watanabe et al. (1973) exposed cats to 0.26, 0.5, and 1.0 ppm ozone for two to 6.5 hours and found increased flow resistance and reduced oxygen exchange capacity. These effects were positively correlated with increased ozone levels. Lung capacity was not affected. Autopsy of the exposed cats showed lung tissue injury severe enough to account for reductions in oxygen exchange capacity.

There is evidence suggesting that ozone may cause destruction of genetic material in living cells. Zelac et al. (1971) exposed Chinese hamsters to 0.24 ozone for five hours and found a significant increase in chromosome breaks in circulating blood lymphocytes. Chromosome breakage also has been found in human cells exposed in laboratory cultures to 9.0 ppm ozone for five to ten minutes (Fetner, 1962). The significance of these studies is not currently know.

Clinical Studies

There is conclusive evidence indicating a relationship between ozone exposure and reductions in human lung functions. Recent studies have shown statistically significant lung function changes in healthy smokers and nonsmokers at concentrations of 0.37 to 0.75 ppm ozone alone and in combination with other pollutants for two hour exposures under intermittent exercise conditions (Bates and Hazucha, 1973; Hazucha et al., 1973). Slight decreases in lung function have been noted in sensitive individuals at 0.25 to 0.37 ppm ozone during light exercise (Hackney et al., 1975). Research by von Nieding et al. (1976) suggests detrimental lung changes may be significant in healthy persons from a two-hour exposure to ozone in the range of 0.15 - 0.25 ppm.

Evidence indicates humans show apparent adaptation to repeated ozone exposures. Hackney et al. (1977a) reported that impairments in lung function from two-hour exposures to 0.5 ppm ozone improved with subsequent exposures. Hackney et al. (1977b) also found that when ozone was administered to Los Angeles and Canadian subjects, the Canadians showed definite decreases in all lung function tests, while the Los Angeles residents did not, apparently indicating the Los Angeles subjects had adapted to higher ozone levels. Such adaptation is not without risk. Freeman et al. (1974) found that rats breathing 0.54 to 0.88 ppm ozone for six months showed a premature aging of the lung (obstructed small airway passages and decreased lung tissue elasticity).

Studies by Hackney et al. (1975) found a four to five hour exposure to 0.5 ppm ozone, 0.3 ppm nitrogen dioxide, and 30 ppm carbon monoxide caused a marked change in the lung function of sensitive individuals. Results of an experiment by Hazucha and Bates (1975) discussed in the section on sulfur oxides and particulates (p. 49) showed an adverse synergistic effect on lung function when ozone and sulfur dioxide were administered together. This experiment indicates that the combination of ozone and other pollutants may affect individuals more strongly than ozone alone.

The most obvious reaction of humans exposed to photochemical pollutants is eye irritation and the sensing of odor. Studies by Henschler et al. (1960) show an odor threshold for ozone below 0.02 ppm. Results also indicated that the odor diminished rapidly following exposure and was no longer perceptible after an average five minute period. The odor persisted longer, an average of 13 minutes, after exposure at 0.05 ppm. In experimental studies, ozone at concentrations ordinarily found in the ambient air of U. S. urban areas has not been found to cause eye irritation (Schuck and Doyle, 1959). Formaldehyde appears to be the constituent contributing most to eye irritation from urban pollution. Acrolein and PAN also may contribute to the irritation (Altshuller, 1978).

Short-Term Epidemiological Studies

The effect of oxidants on the lungs of sensitive persons is not conclusively known, but risks are indicated at the 0.15 to 0.25 ppm ozone level. A study by Schoettlin and Landau (1961) showed that persons with asthma in Los Angeles had significantly more attacks when daily maximum (one hour average) oxidant levels exceeded 0.25 ppm than on days with lower levels.

Short-term oxidant exposures have been associated with increased illness and decreased exercise performance in healthy populations. Increased cough and chest discomfort has been observed in a Los Angeles study of student nurses conducted by Hammer _et al_. (1974) when the maximum hourly oxidant concentrations during the day were 0.25-0.29 ppm. Headaches and eye irritation were found to occur at still lower levels, increasing with oxidant exposure at maximum hourly averages between 0.15 to 0.19 ppm.

Wayne _et al_. (1967) found the performance of high school cross-country runners in Los Angeles was strongly correlated to oxidant levels during the hour before a race. The effects increased proportional to oxidant level increases from 0.05 to 0.03 ppm. Folinsbee (1975, 1977) suggests that decreased performance results from ozone irritating the respiratory system. Irritation from ozone was found to result in a reduction in the respiratory capacity and total volume of the lungs.

Recently, DeLucia and Adams showed in a clinical study (1977) that exercising persons experience chest discomfort and reduction in lung function at a level of 0.15 ppm ozone for a one hour exposure period. These results indicate that exercising individuals have a heightened sensitivity to the adverse health effects of ozone and should be considered a susceptible portion of the population. Table III.E-I summarizes the health effects of ozone.

TABLE III.E-I SUMMARY: HEALTH EFFECTS OF OZONE

Subjects	Concentration (ppm)	Duration of Exposure	Effects	References
Mice	0.07-1.0	3 hrs.	Increased death rate when innoculated with bacterial aerosols (w/ and w/out pre-exposure to cold).	Coffin and Blommer 1970, Miller et al, 1978
Cats	0.26-1.0	2-6.5 hrs.	Increased airway flow resistance and increased lung diffusion capacity correlated with increasing ozone levels.	Watanabe et al, 1973
Adult males	0.15	1 hr.	Decreased lung function and increased respiratory symptoms. Effects enhanced by exercise at 65% maximum.	Delucia and Adams, 1977
Adult males	0.25-0.37	2 hrs.	Threshold for lung function decreases in healthy individuals. Accentuated affects when combined with other pollutants.	Hazucha et al, 1973 Hackney et al, 1975
Asthma patients	0.25	daily mean	Asthma attacks increase in sensitive individuals.	Shoettlin and Landau, 1961
Young athletes	0.05-0.3	1 hr.	Performance related to oxidant levels hour before race.	Wayne et al, 1969
Student nurses	0.15	1 hr.	Increased headache and eye irritation	Hammer et al, 1974

Long-term Epidemiological Studies

Several studies suggest a relationship between oxidant exposure and chronic respiratory illness. Goldsmith and Deane (1965) observed a parallel between high oxidant levels in Los Angeles and persistent cough and phlegm in telephone workers there. Linn (1976) found comparable effects in a similar study on a group of women in Los Angeles. Although both experiments indicate a response to long-term oxidant levels, the study designs did not allow specific oxidant levels to be associated with contribution to respiratory disease. Peters (1973) found that welders continually exposed to 0.01 to 0.36 ppm ozone and 0.01 to 0.08 ppm nitrogen dioxide tended to have a higher incidence of chronic lung disease than nonwelders.

Effects on Agricultural and Other Plants

Injury to vegetation was one of the earliest signs of photochemical oxidant pollution. As early as the 1940s, damage was noted in California plants (Middleton _et al_., 1950). Since these first observations oxidant pollution has become widespread, injuring forests and crop lands throughout California and the eastern United States (Williams _et al_., 1977; Hayes and Skelly, 1977).

In most plants, photochemical pollutants are taken in through the leaves. Ozone and other oxidants enter the leaf through pores (stomata) and then dissolve in the water layer inside the leaf, where they begin to damage the plant (Tingey, 1977).

The initial effect of oxidants on plants is to increase leakage of water and nutrients from internal cells, causing a decrease in photosynthesis and simulating various stress reactions within the plant (Heath, 1975).

Subsequently, the incorporation of nitrogen into plant structures and the transfer of nutrients are affected (Gibson, 1976). Ultimately, disruptions in the internal chemistry of the plant results in either visible or invisible injury.

Visible injury may result from either long-term or short-term exposures. Injury from short-term exposure (no longer than a few hours) usually is visible within 24 hours and is identified by cell collapse and subsequent development of a dead tissue pattern in foliage. The short-term exposures must be relatively high to cause this amount of damage. Other types of injuries usually are associated with long-term or multiple exposures to low pollution concentrations. Such long-term injury manifests itself as a progression of tissue discoloration from yellow to brown and ends with premature leaf drops. Invisible injuries include reductions in growth and yields, and decline in the quality of plant produce. Invisible injury results from both short and long term exposures to photochemical pollutants. Although growth and yield reductions may result from visible damage, the important consideration with invisible injury is that growth and yield can be reduced with no outward indication of damage to the plant.

Short-term Exposures

Experiments investigating effects of one hour ozone exposures consistently have described visible injury to vegetation below 1.0 ppm ozone (NAS, 1976). At levels of 0.2 ppm ozone, Adedipe et al. (1972) reported reduced growth and flower production in four bedding plants. At levels between 0.4-0.25 ppm ozone, Adedipe and Ormrod (1974) found 38-75 percent reductions in radish yields after a 90 minute exposure.

Certain varieties of tobacco have proved to be particularly sensitive to ozone. The tobacco variety Bel W_3 has shown a 48 percent chlorophyll reduction when exposed to 0.3 ppm ozone for two hours (Adedipe 1973). Because of its sensitivity to ozone, Bel W_3 has been used by a number of investigators as a monitor of ozone exposures. The response of this variety of tobacco is often used to determine the frequency and approximate average concentrations of oxidants in the atmosphere (Craker et al., 1974; Heck and Heagle, 1970).

Long-Term Exposures

Oxidants operate in what is called a "cumulative dose-response" manner. This means that plants may be injured over long periods of time from relatively low ozone/oxidant exposures. Even at very low concentrations, the period of exposure is critical in determining the severity of vegetation injury. Long term exposures to low oxidant levels eventually may result in effects as severe as the injury caused by higher short term exposures.

Table III.E-II provides experimental evidence from a number of oxidant exposure studies performed on plant species growing in Montana naturally, or on farms or home garden and greenhouses. The table indicates that many Montana plants show visible injury and reduced photosynthesis, and yield from oxidant concentrations between 0.05 and 0.15 ppm for various periods.

Agricultural plants are sensitive to oxidant pollution. Injury from these pollutants may slow the ripening process and delay harvest times. Tingey et al. (1971) found radishes to be particularly sensitive to ozone. Results from this study noted that at an ozone exposure of 0.05 ppm for eight hours a day, five days a week, for five weeks, radish root weight decreased 54 percent and leaf weight 20 percent. A 30 percent reduction

TABLE III.E-II SUMMARY: OZONE EFFECTS TO SELECTED MONTANA PLANTS

Plant Species	Concentration	Exposure	Response of Vegetation	References
Alfalfa	0.05 ppm	7 hr./day, 68 days	30% leaf dry wt. reduction harvest 1 50% leaf dry wt. reduction harvest 2	Neeley et al. 1977
Alfalfa	0.10 ppm	2 hr./day, 21 days 2 hr./day, 21 days	16% leaf dry wt. reduction 25% leaf dry wt. reduction	Hoffman et al.1975
Wheat, cultivar	0.20 ppm	4 hr./day, 7 days (anthesis)	30% yield loss	Shannon and Mulchi, 1974
Soybean	0.05 ppm	8 hr./day, 5 day/wk. 21 days 8 hr./day, 5 day/wk. (mixture of O_3 & SO_2 for same period)	13% leaf injury 16% leaf injury 20% root dry wt. reduction	Tingey et al. 1973
Corn, sweet, cultivar-Golden Midget	0.05 ppm	6 hr./day, 64 days	9% kernel dry wt. reduction	Heagle et al. 1972
Bean, cultivar	0.13 ppm	8 hr./day, 28 days	79% leaf fresh wt. reduction 73% root fresh wt. reduction 70% height reduction	Manning et al. 1971
Bean, cultivar	0.15 ppm	2 hr./day, 63 days	33% biomass reduction 46% pod fresh wt. reduction	Hoffman et al. 1973
Bean, cultivar	0.15 ppm 0.15 ppm 0.15 ppm 0.15 ppm	2 hr./day, 14 days 3 hr./day, 14 days 4 hr./day, 14 days 6 hr./day, 14 days	8% leaf dry wt. reduction 8% leaf dry wt. reduction 20% leaf dry wt. reduction 49% leaf dry wt. reduction	Maas et al. 1973
Pinto beans	0.06 ppm	5 hr./day, 5 days/wk. 40 days	48% top wt. reduction 25% height reduction	Manning, 1978
Potatoes 4	0.05 ppm 0.05 ppm	730 days 326-533 days	34-50% yield loss 20-26% yield loss	Heggestad, 1973
Potato cv.	0.15 ppm	3 days	95% leaf injury	Brasher et al.1972
Radish	0.05 ppm 0.05 ppm	8 hr./day, 5 days/wk. 35 days 8 hr./day, 5 days/wk. (mixture of O_3 & SO_2)	54% root fresh wt. reduction 20% leaf fresh wt. reduction 63% root fresh wt. reduction	Tingey et al. 1971
Ponderosa pine	0.15 ppm 0.15 ppm	9 hr./day, 10 days 9 hr./day, 20 days	4% photosynthesis reduction 25% photosynthesis reduction	Miller et al. 1969
Ponderosa pine	0.15 ppm 0.15 ppm	9 hr./day, 30 days 9 hr./day, 60 days	25% photosynthesis reduction 34% photosynthesis reduction	
Ponderosa pine	0.10 ppm	6 hr./day, 126 days	12% root length reduction 21% stem dry wt. reduction 26% root dry wt. reduction	Wilhour & Neeley, 1977
Western pine	0.10 ppm	6 hr./day, 126 days	13% leaf dry wt. reduction 9% stem dry wt. reduction	
Poplar, hybrid	0.15 ppm	8 hr./day, 5 days/wk. 42 days	50% shoot dry wt. reduction 56% leaf dry wt. reduction 47% root dry wt. reduction	Jensen & Dochinger, 1974
14 plants from an Aspen forest	0.15-0.30 ppm	3 hr./day, 5 days/wk. through the growing season	significant amounts of leaf injury and losses in plant wt. and seed wt.	Haward & Treshow, 1975
Petunia	0.05-0.07	24 hr./day, 53 days	30% flower fresh wt. reduction	Craker, 1972
Carnation	0.05-0.09 ppm	24 hr./day, 90 days	50% reduced flowering (reduced growth)	Feder, 1970

in wheat yields occurred when plants were exposed during flower formation (anthesis) to 0.2 ppm ozone for four hours daily (Shannon and Mulchi, 1974).

Heagle *et al.* (1972, 1974) found a reduction in the yield of sweet corn and soybeans after 0.10 ppm ozone for six hours a day over a growing season. They suggest the threshold level for measurable effects is between 0.05 and 0.10 ppm ozone throughout the growing season. These levels are in the range of growing season ozone averages in the eastern United States.

Ozone also affects vegetation indirectly through its effects in other organisms plants need for optimal growth. Recent studies have noted growth losses in a number of experimental plants from ozone-caused reductions in nitrogen-fixing bacteria associated with plant roots. The level necessary to cause significant loss in height and dry weights of above ground portions of pinto bean plants through bacteria reductions was 0.06 ppm ozone for 40 days (Manning, 1978).

Plants native to Montana also appear sensitive to low level ozone exposures. Howard and Treshow (1975) exposed 14 species of an aspen plant community to ambient air containing 0.05-0.07 ppm ozone and to ozone levels between 0.15 and 0.30 for three hours per day, five days per week, for an entire growing season. Foliar injury was noted in all species and reduced growth and seed production was found at elevated ozone levels in most cases. Two dominant timber species in Montana, ponderosa pine and western white pine are sensitive to ozone. Wilhour and Neely (1977) found that exposure of ponderosa pine to 0.10 ppm ozone for six hours per day, seven days per week, for 18 weeks caused reductions in root length and dry weight of the roots and stem. They also noted reductions in dry weight of the stem and foliage in western white pine at these concentrations and exposure durations.

Exposure to PAN and Hydrocarbons

PAN as a constituent of the photochemical oxidant mix can injure vegetation. The type of damage caused by PAN is visibly different from ozone damage for most high level, short-term exposures (EPA, 1976). The lowest levels of PAN found to impact agricultural plants in controlled exposure experiments are 0.20 ppm for 0.5 hours, 0.10 ppm for one hour, and 0.04 ppm for four hour exposures (Jacobson, 1977).

Among hydrocarbons, ethylene has the greatest potential for adverse impact on agricultural and native plant communities. It is a plant growth regulator under natural conditions and is harmful to plants at levels above those normally found in nature. Experimental exposure to ethylene concentrations as low as 0.005 ppm (for 24 hours) may have a harmful effect on plant growth and development. Agricultural crops such as squash, beans and peas are sensitive to ethylene damage.

Pollutant Interactions

Under ordinary field conditions, plants are often exposed to a combination of pollutants. Experiments analyzing visible injury and growth/yield reductions caused by exposures with combinations of pollutants more accurately represent real life situations.

Studies on ozone and sulfur dioxide indicate that synergistics effects sometimes result when they are combined. Very small amounts of ozone and sulfur dioxide in combination can damage vegetation. Adverse effects have been noted at exposures of 0.1 ppm ozone with 0.3 ppm sulfur dioxide over a one hour exposure in alfalfa (Bennett and Hill, 1974) and 0.05 ppm ozone with 0.05 ppm sulfur dioxide for four hours in eastern white pine (Costonis, 1973). Effects have been observed at ozone levels as low as 0.008 to 0.01 ppm with sulfur dioxide levels from 0.02 to 0.03 ppm in peanuts (a relative of alfalfa and beans) for a two hour exposure (Applegate and Durrant, 1969).

Other pollutants in combination with ozone also have been found to damage vegetation at low concentrations. Experiments by Kress and Skelly (1977) show that 0.05 ppm ozone and 0.14 ppm sulfur dioxide together caused visible injury and decreased growth to sycamore and loblolly pine at an exposure duration of six hours a day for 28 consecutive days. These researchers also noted that combinations of 0.05 ppm ozone, 0.14 ppm sulfur dioxide, and 0.01 ppm nitrogen dioxide resulted in significantly less grwoth than in control plants which were not exposed to pollutants. Other pollutant combinations did not produce comparable growth reductions.

One study on the effects of ozone and nonmethane hydrocarbons (hexane) compared visible injury with growth and chlorophyll content in pinto bean plants. The results suggested that visible injury was not a good predictor of growth losses (Todd and Arnold, 1961), Investigations on the combined effect of PAN and ozone on vegetation have not been definitive. More work in this area is essential.

Effects on Livestock and Other Animals

No direct effects on domestic animals and wildlife have been found. Laboratory animals studies are discussed in the human health section of this section.

Effects on Ecosystems

During the last 20 to 25 years, photochemical oxidants have injured both natural and agricultural ecosystems. Oxidants increase the susceptibility of plants to insects and disease. Long-term low level oxidant pollution is further linked with crop reductions and a variety of impacts in natural ecosystems (EPA, 1977).

In general, oxidant damage to crops is most dramatic in the short term because they are simple, usually consisting of a single plant species with little resilience under pollution stress. Oxidant damage to crops often is immediate and sometimes catastrophic. Studies by Thompson (1968, 1970) have shown that oxidant stress reduces photosynthesis and water use by plants, increases premature leaf and fruit drop, and reduces the marketability of fruit products.

Natural ecosystems are structurally complex, relatively stable associations with living and nonliving components interacting in equilibrium. The effects of oxidant pollution on such systems are complex, with most still not completely understood. In general, it is known that disturbances by oxidant pollution may return natural ecosystems to a simpler stage in structural and functional development. There may be a selective removal of taller (overstory) vegetation in favor of lower growing forms; a shift from forests toward shrubs and herbs (Woodwell, 1970).

Structural changes in natural ecosystems have been found to be either directly or indirectly related to oxidant impact. Wert (1969) found, using aerial photography, that 46,230 acres of a total 160,950 acres of ponderosa-Jeffrey pine forests in the San Bernardino Mountains 80 miles downwind from Los Angeles had severe damage from oxidant pollution. The researchers hypothesize that such damage eventually would result in the replacement of pollutant sensitive ponderosa and Jeffrey pine by more resistant but less economically and aesthetically desirable trees such as incense cedar. Openings in the forest's overstory would also cause changes in the amount of light, water, and other growth essentials available to shorter plant species, possibly altering their environment (EPA, 1977).

In a series of controlled exposure experiments, Treshow and Stewart (1973) reinforced the observations made in the San Bernardino studies. They exposed over 70 species native to grass-oak and aspen-conifer plant communities for two hours to 0.15, 0.25, 0.30, and 0.40 ppm ozone. Results indicated that aspen is sensitive to ozone at all those levels.

The researchers considered the sensitivity of aspen to be very significant. Aspen is vital to the aspen-conifer plant community in that it provides essential shade for young white fir seedlings and other plant species. It also was found that aspen acts as a filter, keeping high ozone concentrations from reaching ground level.Oxidant levels in southern California forests often are greater than the concentrations used by Treshow and Stewart and are considered to have caused fundamental structural changes in the ponderosa-Jeffrey pine ecosystems located there (Miller et al., 1972; Williams et al., 1977).

In altering the vegetation types in natural ecosystems, oxidant pollution indirectly affects all components of the system. Modifications in consumer populations result from disturbances in food availabiility. Studies (Stark, et al., 1968; Stark and Cobb, 1969) have demonstrated that ponderosa pine weakened by oxidant injury is subject to increased attach by pine bark beetles. It is known that bark beetles accelerate the species shifts initiated in the forest communities stressed by oxidant exposure.

Such exposure also affects the portion of the forest ecosystem responsible for decomposition. Small animals, insects, fungi, and bacteria that depend on forest floor litter as a food source will decrease parallel with a decrease in total productivity of the forest ecosystems (Woodwell, 1970).

Small changes brought on by oxidant exposure to natural ecosystems impact and magnify through all levels of the system. As a result of these changes it may take many years for the system to reach a new equilibrium, which sometimes may occur only after the replacement of the former system with an entirely different one.

Effects on Materials

Ozone and other photochemical oxidants contribute to the degradation of materials. Researchers have identified ozone exposure with accelerated aging in a wide variety of economically important meterials, including rubber, textile dyes and fibers, and cetrain paints. Data on the effect of ozone on materials are summarized in Table III.E-III.

TABLE III.E-III

SUMMARY: MATERIAL EFFECTS OF OZONE

MATERIAL	RESPONSE	EXPOSURE DURATION HR.	CONCENTRATION PPM	REFERENCE
Rubber	Breakage	150-250 400-500 500-700	0.048 (Summer) 0.042 (Falls) 0.024 (Winter)	Meyer and Sommer, 1957
Cotton (wet)	Fluidity change	1200	0.02-0.06	Bogatey *et al.*, 1952
Cotton (dry)	No Response	1200	0.02-0.06	
House Paint; Electrical Coil Coating; Automotive paint; Industrial Maintenance Coating	Increased Deterioration	400	1 ppm, 66 C, 70% relative humidity; followed by 1 hr. darkness 49 C, 100% relative humidity	Campbell et *al.*, 1974

Rubber compounds exposed during the summer appear the most sensitive, showing detrimental effects at levels near natural background. The deterioration rates of paint increased significantly with exposure to 1 ppm ozone. The relatively sensitive paints tested were: oil-based house paint, industrial maintenance paint, latex, coatings on electrical coils, and automotive paint (Campbell et al., 1974). No dose-response data are available for dyes although it has been shown that dyes are sensitive to ozone damage particularly if sulfur dioxides or nitrogen dioxides also are present. Garments treated with sensitive dyes have been known to fade within ten days (Salvin, 1969).

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III.F. VISIBILITY

". . . this giant spread of land, this plain . . . Everywhere but to the mountained west it flowed forever. Farther than a man could think, beyond buttes blued by distance, floating in it, the earth line lipped the sky. And hardly anything,. . to see . . . just emptiness and open sky. Air like tonic, days like unclaimed gold. . . Grass beyond the earth line, which wasn't any line but just the farthest reach of eye. World without end . . . ."(Guthrie, 1956).

The impairment of visibility in the atmosphere is caused primarily by small particles which scatter and absorb light. Larger particles and colored gases, such as nitrogen dioxide, also reduce visibility, but to a lesser degree. Reduced visibility will be noticed both as a decrease in how far things can be seen clearly and by a color change in the objects viewed.

When light rays strike a particle they may be absorbed or deflected into a new direction (scattered). Scattering is particularly strong when the wavelength of the light is approximately the same as the radius of the particle. The different elements that determine the visibility of an object are shown in Figure III.F-I. In this case, the distant mountain is seen against the sky.

Both sky and mountain are illuminated by the sun but there is a distinct difference in their color and brightness. This contrast between the brightness of the object and the background determines the degree of visibility. Extreme contrast is not always necessary to distinguish an object. A tree can be seen against a hillside if it is close enough, but a tree on the horizon can be seen much farther. Most people can identify a nondescript object when the contrast between the relative brightness of the object and the background is between two percent and six percent. The minimum contrast at which an object can be perceived is called the contrast threshold. The distance at which it becomes impossible to see an object is called the "visual range."

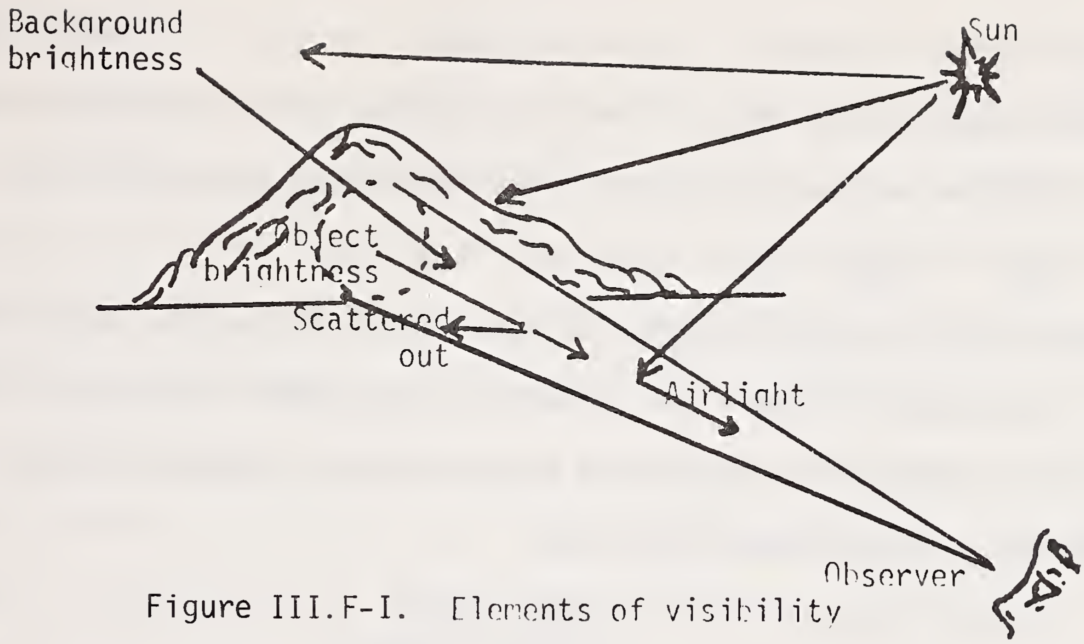

Figure III.F-I. Elements of visibility

As light coming from the mountain is scattered, less of it reaches the observer. The greater the distance between the observer and the mountain, the greater will be the number of particles in the way.

Second, other light scattered into the sight path of the observer further decreases the contrast, thereby reducing visibility. Light from the sun coming directly or reflected from elsewhere may scatter off particles in the air between the observer and the object.

The amount of light scattered depends on the number, size and chemical composition of the particles in the sight path. For any group of particles it is possible to measure a "scattering coefficient," or the scattering ability of the particles expressed in terms of the scattering potential of a specific length of sight path, such as one meter. This is abbreviated "b_{sp}" or b-scattering particle, where "b" is an arbitrary symbol for the scattering coefficient. The absorption of light by the particles is similarly measured and is abbreviated "b_{ap}." The scattering power of gas molecules in the air is constant at constant atmospheric density. It is called Rayleigh scattering, in honor of Lord Rayleigh (a 19th century English physicist) and is abbreviated "b_{Rg}." Some gases in the air, such as nitrogen dioxide, can absorb visible light, a process abbreviated "b_{ag}." The sum of these four terms measures the total light reduction per meter, and is called the extinction coefficient, abbreviated "b_{ext}" ($b_{ext} = b_{sp} + b_{ap} + b_{Rg} + b_{ag}$).

It is possible to write a mathematical equation relating the extinction coefficient to the visual range if we assume that the object viewed is a flat black color and the contrast threshold is known (Duntley, 1948). When the contrast threshold is two percent, then the

$$\text{visual range in miles} = \frac{2.44 \times 10^{-3}*}{b_{ext}\ (\text{per meter})}.$$

The natural scattering coefficient of clean air (b_{Rg}) is approximately 1.5×10^{-5} per meter. If there were no absorption or scattering by particles and no absorption by gases, then $b_{ext} = b_{Rg}$. If we divide 1.5×10^{-5} per meter into 2.44×10^{-3}, we find that the natural light scatter by atmospheric gases limits visibility to about 160 miles. If particles are added to the clean air, the visibility will decrease according to the scattering power of the particles ($b_{ext} = b_{Rg} + b_{sp}$). For example, if the particle scattering coefficient is 6×10^{-5} per meter, the extinction coefficient is 7.5×10^{-5} per meter and the visual range is about 40 miles.

Visibilities measured in urban areas frequently are between 20 and 50 miles. Under conditions of extreme pollution, the visual range may drop to less than a mile. In dry western rural locations a visual range to 65 to 85 miles is common, with perhaps ten percent of the days having a visibility above 90 miles.

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*The "exponential notation" is useful for expressing very small numbers. For example, 1.5×10^{-5} is the same as 0.000015 and 1.5×10^{-4} is the same as 0.00015. Thus expressed, these small numbers are much less ambiguous to read aloud than "fifteen hundred thousandths."

A view may be severely degraded even when objects are visible to some degree. The appearance of an object three miles away will be distinctly altered when the visual range is thirty miles. Long before an object disappears into the haze, it will lose much of the "crispness" of its appearance. Subtle shifts in the color of the scene will be observed even when it's well within visual range. A dark object can appear quite grey when the visibility is still 100 miles. The brown color of a pollution haze may be due more to the color of light scattered from particles than to any colored gas such as nitrogen dioxide (Waggoner, 1977).

Fine particles between 0.1 and 1 micrometer are significantly more effective in scattering light than the same mass of larger or smaller particles. It would require 20 ug/m^3 of particles five micrometers in diameter to scatter the amount of light scattered by one ug/m^3 of particles one micrometer in diameter. This is because the wavelength of visible light is approximately 0.5 micrometers. Thus large particles, such as those from road dust, reduce visibility only when they are present in large amounts. Sulfates and other particles formed from gas in the atmosphere are concentrated in the size range between 0.1 and 1 micron. In a recent study in Phoenix, 44 percent of the light scatter was found to be due to sulfates, 31 percent to nitrates and photochemical aerosol, 17 percent to Rayleigh scatter, and eight percent to other particles. At the Grand Canyon of the Colorado, 43 percent of the scatter was natural Rayleigh scatter, while 36 was from sulfates, nine percent from nitrates and photochemical aerosol, and 12 percent due from other particles (Trijonis and Yuan,1978).

There is no fixed ratio between the total mass of particles in the air and the amount of visibility degradation. Particles grow as they absorb water so the number of particles in the most sensitive size range can change dramatically as the relative humidity rises, producing marked reductions in visibility. Because light scattering is dominated by fine particles while the bulk of particulate is coarse particles, it is possible to change visibility dramatically by changing the relative amounts of fine and coarse particles in a given mass of particulates. This is why an air quality standard for total suspended particulate will not protect against visibility degradation.

Changes in visibility also affect the view of the night sky. As the number of airborne particles increases, the dimmer stars become more faint and many will disappear. The drama of the night sky is one of the most frequently remarked memories of visitors to Montana. An extinction coefficient of 3×10^{-4} per meter would obscure stars dimmer than the 5th magnitude, on the average, thus removing more than two-thirds of the stars from view (Leonard et al., 1977).

Suspended particulate deflects light from the direct beam of the sun. A national network has for many years measured atmospheric opacity (turbidity) caused by particles in the air. The data shows a trend of increasing turbidity over the past half century. Data from Montana indicates that over the past few years up to 17 percent of direct sunlight has been scattered out of direct beam of the sun (Flowers et al., 1969). An even higher percentage of ultra-violet light, vital to the growth of plants, is scattered out of the direct beam. Bolin and Charlson (1976) have suggested sulfate aerosol from industrial emissions is the primary cause of the increasing turbidity, although others have attributed it to a natural variation of cloudiness (Angell and Korshover, 1978).

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III.G. FLUORIDE

Introduction

Particulate and gaseous fluoride is emitted from many industrial facilities including aluminum smelters, phosphorous plants, and fossil-fueled generators. High ambient concentrations have been measured near the Anaconda aluminum smelter at Columbia Falls, and the Stauffer Chemical Company phosphorous plant at Ramsay. Additional information on the sources and ambient measurements of airborne fluoride in Montana can be found in Section II.B and Appendix A.

There is no evidence that human health is being affected at ambient fluoride concentrations found in Montana.

Air and vegetation fluoride levels commonly recorded in Montana have been shown to adversely affect plants and animals. Fluoride has been shown to cause alterations in plant biological processes leading to tissue death and growth losses in many types of plants, particularly timber species. Herbivorous animals consuming fluoride contaminated vegetation show bone abnormalities and reductions in vital biological functions. Insects, particularly honeybees are sensitive to fluoride accumulation.

The persistence of fluoride in the environment leads to accumulation and extensive effects in ecosystems. Fluoride has been observed to accumulate in food chain segments of Montana ecosystems.

Human Health Effects

Industrial fluoride emissions in the ambient air can be hazardous to human health. Steinegger (1969) reported skin sore developed on persons living near an aluminum smelter in Italy. Diagnosis indicated the sores resulted from excessive fluoride exposure. Columbini *et al*. (1969)

reported similar sores on residents living in the vicinity of an aluminum smelter where ambient gaseous fluoride levels approached and sometimes exceeded 12.2 ppb. Particulate fluoride also was high in the area.

A variety of maladies found in residents living near fluoride emitting industries is collectively referred to as "neighborhood fluorosis." The disorders include arthritis in the muscle-skeleton system, sores on the skin, and many other effects on the gastro-intestinal tract and respiratory system (Waldbott, 1970). Neighborhood fluorosis is a documented result of exposure to elevated fluoride levels in the air, soil, water, and food near emission sources. There are a number of other studies showing decreased hemoglobin levels in residents inhabiting similar areas with elevated air fluoride (Balazova *et al*. 1970; Leloczky, 1971).

A National Academy of Sciences (NAS, 1971) publication reported that occupational exposures are a major cause of fluoride related health problems. Carnow and Conibear (1977), in a study of aluminum smelter workers noted significant decreases in lung function and an increased incidence of possible bone abnormalities in groups of employees exposed to elevated pollution levels. The study design did not allow determination of which health problems were caused by which pollutants in the smelter. Hodge and Smith (1977) said a total fluoride level of 3000 ppb for an eight hour exposure per day caused no short-term health effects.

The presence of fluoride in air, water, and food results in lifetime accumulation of the substance in humans (defined as total fluoride body burden). Approximately 96 to 98 percent of fluoride retained by the body is stored in the bones and teeth (NAS, 1971). Increases in bone hardness and density can be detected at bone fluoride concentrations of 4000 ppm, measured on a defatted dry bone weight basis (Waldbott, 1970). The physiological and health implications of elevated bone fluoride and bone hardness and density are not well understood. It is known, however, that dental health is improved by increased tooth hardness resulting from low levels of fluoride in teeth. Excessive levels of fluoride during tooth formation causes tooth staining and malformations (NAS, 1971).

The Illinois Institute of Environmental Quality (1973) in formulating an ambient air quality standard determined that the most important health consequence of fluoride resulted from the total amount of fluoride accumulated in the body. This study found that normal breathing of air containing 0.85 ppb total fluoride resulted in only two percent or 0.01 milligrams of the total 0.689 milligrams daily fluoride intake coming from the air, with food borne fluoride contributing 25.4 percent and water 72.6 percent. These results assumed that fluoride in the food and water came from uncontaminated sources. The researchers concluded, based on these assumptions, that 0.85 ppb air fluoride would not contribute to excessively high bone fluoride levels, even when exposure was continued for 30 years.

Consumption of food and water contaminated by fluoride results in increases in the fluoride body burden. Tsunoda _et al_. (1972) found that urinary fluoride excreted by men and women consuming locally grown food over a 24 hour period was approximately two milligrams higher in polluted areas than in nonpolluted areas. If it is assumed that 50 percent of the ingested fluoride is excreted (Rose and Marier, 1977) then these data suggest the intake of two milligrams per day resulted directly or indirectly from airborne fluorides. The authors do not indicate an airborne fluoride level for the two study areas.

Rippel _et al_. (1967) studied the dietary intake of fluoride in children from areas with and without fluoride air pollution. The researchers found that children from polluted areas had a daily fluoride intake of 2.15 milligrams, compared to 1.01 milligrams for children from nonpolluted areas. Of this total, 25 percent or 0.55 milligrams was from the air in the contaminated area and 1 percent of 0.01 milligrams came from air in the control area. Airborne fluoride appeared to contaminate the locally grown fruits and vegetables in the polluted area, contributing substantial fluoride to the diet. Children from polluted areas acquired 1.05 milligrams from food contaminated by airborne fluoride, compared to only 0.48 milligrams taken from the same source by children from nonpolluted areas. The results from these studies emphasize the indirect contribution of airborne fluoride on the accumulation of fluoride in the body in contaminated environments.

Effects on Crops and Other Plants

Fluoride* toxicity to vegetation (phytotoxicity) was documented first in 1850 by Stockhart and Schloder in Europe (Weinstein, 1977). Since then there has been much scientific documentation of damage to plants by fluoride emitted from copper smelters, brick works, glass factories, phosphorous and phosphate plants, and aluminum reduction facilities. The aggregate of data from these studies shows fluoride is the most phytotoxic of all the common air pollutants, being over 50 times more injurious to vegetation than either sulfur dioxide or the ozone/oxidant complex (Weinstein, 1977).

Fluoride is taken up by plants from air, soil, and water. Fluoride phytotoxicity usually is associated with gaseous fluorides from the atmosphere. Airborne particulate fluoride and soil/water fluoride in general are not taken up in quantities sufficient to injure plants, but may accumulate on vegetative surfaces and lead to adverse effects in foraging animals consuming the contaminated material (NAS, 1971).

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*Fluorides considered in the vegetation impacts section are all gaseous. Evidence shows that the phytotoxicity of different gaseous fluorides is equivalent. Particulate forms of fluoride will be mentioned explicitly when applicable. All weights and concentrations of fluoride are on a dry weight basis unless otherwise noted and micrograms per gram (ug/g) and parts per million (ppm) are equivalent.

Airborne fluoride enters the plant through the stomata, is dissolved in the internal leaf tissues, and is ultimately transferred to the leaf edges where it accumulates (Jacobson _et al_. 1966; Tourangeau _et al_. 1977). Fluoride affects many biological plant functions. A variety of biological processes such as photosynthesis, carbohydrate utilization, starch synthesis or growth, oxygen uptake, and cell wall development are adversely affected by fluoride (Chang, 1975). The threshold for each of these effects occurs at a different level in each plant species, with further variation among different species. In general, visible injury and/or growth inhibition occurs in plants when continued fluoride exposure causes changes in plant metabolism (Weinstein, 1977).

A darkening or water soaked appearance of plant leaves is the first sign of visible injury from fluoride exposure in broad leaved plant species. A loss of chlorophyll (chlorosis) is an early indication of visible injury in conifers. The onset of both darkening and chlorosis of leaves occurs at leaf edges, where the highest concentration of fluoride is, and proceeds toward the midvein in broad leaves or the base in conifer needles. These forms of visible injury are generally followed by death of portions of the leaf surface (necrosis). Chlorosis, necrosis, or a darkening of the leaf surface can occur from a short-term high level exposure or longer low level exposures (EPA, 1976). Tissue disruptions in the internal structure of plant leaves have been associated with fluoride-caused tip and edge necrosis in narrow and broad-leaved plants. Such disruptions indicate fluoride's impact on plants and are distinguishable from tissue changes caused by winter injury, drought or natural aging of the plant (Carlson, 1978; Solberg and Adams, 1956).

Growth and yield losses in plant populations result from airborne fluoride contamination. These effects may occur even when there is no visible symptom of fluoride injury. Treshow et al. (1967) found that fluoride exposure caused significant growth reduction in Douglas fir without characteristic fluoride type visible injury. There is also evidence that fluoride sometimes stimulates plant growth. These effects, however, usually result in a spindly or twiggy growth not beneficial to plants (Weinstein, 1977).

Accumulation of Fluoride and Plant Response

Fluoride is unusual among air pollutants in its ability to accumulate in plants. Natural or "background" levels of fluoride in plants come from soil and water fluoride. Abnormally high fluoride levels in plants usually indicate they have been subjected to a fluoride-contaminated atmosphere.

Plant populations in areas without airborne fluorides normally have low levels of fluoride in their foliage. Montana studies by Kay et al.(1975b)found natural fluoride levels of 4.5 ppm in grasses and 2.8 ppm in conifers. Gordon et al. (1976) showed eastern Montana ponderosa pine to have less than 2 ppm in 1, 2, 3, and 4-year-old needles. Carlson et al. (1974) found 0-2.7 ppm fluoride in 1 and 2-year-old needles of western Montana lodgepole pine. Uncontaminated alfalfa from throughout the U.S. was found to have an average fluoride level of 3.6 ppm (Suttie, 1969a).

The amount of plant fluoride accumulated from contaminated environments is variable, and depends on the dosage (concentration and time of exposure), plant susceptibility, and environmental conditions. Field studies near fertilizer plants and/or aluminum smelters in Washington (Shaw et al. 1951), Oregon (Compton, 1960), and Canada (Linzon, 1971; Sidhu, 1978) have shown that plant tissue can accumulate up to 1270 ppm fluoride, approximately 140 times greater than uncontaminated tissue.

Fluoride accumulation in plants also depends in part on their type and surface area. Knabe (1969) showed that fluoride accumulation increases with height of vegetation. Gordon *et al*. (1976) has illustrated the "scavenging" properties of taller growth forms. In addition, Gordon and Tourangeau (1977) showed that near fluoride sources the side of the plant facing the source acquires more fluoride than the opposite side.

Broadleaf and deciduous conifers accumulate fluoride throughout the growing season and then cast their foliage in the fall. These plants, therefore, do not accumulate significant amounts of fluoride for more than one growing season. Only small amounts of fluoride remain in these plants for more than one year. Evergreens, on the other hand, retain foliage for four or more years under normal conditions and accumulate fluoride throughout the period of foliage retention. Sidhu (1978) found that conifer needles accumulate almost all they will accumulate by the end of the second growing season. From these results, Sidhu hypothesized that the full impact of fluoride accumulation in these needles is not felt until after two years of growth. He said an air quality standard should be based on two years accumulation in evergreens.

The level of fluoride accumulated in vegetation is proportional to the exposure period and concentration of gaseous fluoride (usually hydrogen fluoride). This relationship is described by the dose rate equation:

$$F = K \cdot C \cdot T$$

where F represents the concentration of tissue fluoride after exposure and correction for natural levels, C equals the average concentration of atmospheric fluoride in parts per billion, T is the exposure time in days, and the accumulation coefficient (K) is the amount of fluoride accumulated by a given species at a given concentration in air per day.

Data from exposure studies (Guderian _et al_. 1969; Benedict _et al_. 1964; Jacobson, 1966) allows determination of accumulation coefficients for various plant species. The research indicates that the accumulation coefficients can predict the degree of fluoride accumulation in vegetation over given periods with rapid accumulation reflected by high coefficient numbers. Table III.G-I indicates accumulation coefficients for a number of important forage plants in Montana. Data from Table III.G-I strongly suggests that relating ambient air concentrations to tissue fluoride is possible over a range of atmospheric concentrations. Table III.G-I shows ranges of air fluoride concentrations that would result in 30 or 40 ppm fluoride in several Montana forage species if they were exposed continuously to the given levels for 30 and 150 day periods. The figures were determined with the accumulation coefficient of each species.

The data used to formulate the dose rate equation are based on controlled exposure studies and relate only to the effects of hydrogen fluoride (HF). Comparisons of results between laboratory exposure studies and field experiments are difficult because of the many additional variables influencing field studies, such as the various fluoride pollutants, weather, soil, growth, and frequency of pollutant episodes. Nonetheless, Israel (1974) found in a field study that accumulation coefficients were approximately 3.8 in orchard grass and perennial ryegrass, twice as high as in laboratory studies. A similar study conducted by Davison and Blakemore (1976) also found that plants under field conditions accumulated fluoride faster than in laboratory studies. These findings showed that gaseous forms of fluoride were the most

TABLE III.G-I

Accumulation Coefficients and Projected Fluoride Concentration Ranges (ppb) Which will Result in the Accumulation of 30 or 40 ppm Tissue Fluoride over 30 and 150 days in Various Montana Forage Crops.

Crop	Accumulation Coefficient	30 days		150 days	
		30 ppm	40 ppm	30 ppm	40 ppm
Adapted From Guderian (1969)					
Italian Ryegrass	3.48 ± 2.14	0.18-0.74	0.23-0.99	0.10-0.15	0.05-0.20
Perennial Ryegrass	3.13 ± 1.56	0.23-0.54	0.31-0.72	0.05-0.11	0.06-0.15
Orchard Grass	1.74 ± 0.13	0.54-0.62	0.71-0.83	0.11-0.12	0.15-0.17
Timothy	1.93 ± 1.29	0.32-1.56	0.41-2.09	0.06-0.32	0.09-0.41
Alfalfa	2.09 ± 0.64	0.36-0.68	0.49-0.92	0.07-0.13	0.10-0.18
Red Clover	3.13 ± 1.83	0.21-0.77	0.27-1.02	0.10-0.16	0.05-0.21
White Clover	5.00 ± 3.00	0.12-0.50	0.17-0.67	0.02-0.10	0.04-0.13
Alaska Clover	3.03 ± 1.46	0.22-0.63	0.29-0.73	0.05-0.12	0.06-0.17
Adapted from Israel (1974)					
Perennial Ryegrass and Orchard Grass	4.64 ± 0.98	0.18-0.27	0.24-0.36	0.04-0.05	0.05-0.07

important contributor of both internally and externally retained plant fluoride. A more rapid uptake of airborne fluoride by plants in the field was postulated as a cause of the differences between laboratory and field results.

Near the Anaconda Aluminum Company (AAC) smelter at Columbia Falls, Montana, Carlson and Dewey (1971) found an average tissue fluoride concentration in grasses, forbs, shrubs, and conifers of 34 ppm after a five month exposure to levels between 0.16 and 0.21 ppb.* At atmospheric fluoride levels between 0.13 and 0.16 ppb, vegetation accumulated 16 ppm fluoride. Carlson and Dewey noted injury to vegetation at both these levels and concluded that the aluminum smelter was the source of elevated fluoride and resultant vegetation damage around the facility.

Accumulation of tissue fluoride has been associated with changes in biological function, visible injury, and growth/yield losses in vegetation. The percentage of fruit set, annual shoot growth, number of buds, and number of spurs in cherry trees was shown to be adversely affected at an average fluoride content of 6 to 10 ppm (Facteau and Mellenthin, 1976). Woltz (1964) found photosynthesis in Gladiolus leaves was significantly reduced when tissue fluoride content exceeded 26 ppm. Similarly, Applegate and Adams (1960) found that 10 ppm fluoride in bean leaves caused a reduction in photosynthesis. McLaughlin and Barnes (1975) showed that fluoride accumulation of 10 ppm or less in pines and 60 ppm in hardwoods caused reduced photosynthesis in the pines and stimulated respiration in the hardwoods. The researchers interpreted these findings as an indication that low fluoride levels may cause growth reductions in trees.

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*Air values calculated from correlation between continuous fluoride monitors and static fluoride samplers established by the EPA during the studies (EPA, 1974).

Growth reduction is here defined as an irreversible decrease in mass, or a decrease in diameter, length, area, or volume (Weinstein, 1977). The weight of alfalfa, orchard grass, and romaine lettuce (Benedict et al. 1964) leaf area and size of citrus (Brewer et al. 1960); Leonard and Graves, 1966) and weight of roots (Benedict et al. 1964) are all reduced by fluoride accumulation at various levels. Decreases in diameter growth of ponderosa pine (Lynch, 1951), mixed conifer forest species (Carlson, 1978), and Hemlock-Cedar forest species (Bunce, 1978) all have been documented to result from fluoride accumulation in conifer tissue. Treshow et al. (1967) found a significant decrease in the increments of annual growth of Douglas fir at an average fluoride level of 150 ppm in five year's needles.

Carlson (1978) showed that near the Anaconda aluminum plant, an average fluoride content less than 30 ppm in mixed conifer foliage over a 10 year period correlated with a 19 percent diameter growth reduction. A ten year average fluoride level of less than 100 ppm reduced diameter growth 35 percent. Work by Williams and McAllister (1978) supports Carlson's findings and shows that individual years' diameter growth of lodgepole pine in the area of Carlson's study were reduced proportionally to AAC fluoride emissions over a twenty year period. These results were found to be independent of tree age, environmental conditions and plant community composition.

Growth reduction can also be described as a reduction in the volume or productivity within forest ecosystems. Carlson (1978) in Montana, and Bunce (1978) in British Columbia quantified volume reduction caused by fluoride contamination and indicated considerable economic loss from decreased timber yields. Carlson (1978) found that fluoride killed trees and reduced timber volume worth more than $67,000 over a ten-year period. For diameter growth reductions alone, Bunce (1978) found fluoride damage worth $90,000 over twenty years.

A number of plant species are susceptible to visible injury at low foliar fluoride levels. Hitchcock et al. (1962) found injury threshold levels as low as 12 ppm fluoride in gladiolus. Carlson (1978) found that low fluoride levels in conifers affected a number of needle conditions related to the health of the trees. Of the three conifers studied, western white pine was most sensitive, showing an increase in needle abnormalities (pathologies) when fluoride content was as low as 8 ppm, or even less. Both lodgepole pine and Douglas fir showed increases in needle pathologies between 12 and 15 ppm tissue fluoride. Carlson (1978) states that adverse effects to conifer needles begin at concentrations slightly above natural fluoride levels.

The work of Sidhu (1978) supports Carlson's findings. He showed, in a field study near a Newfoundland phosphorous plant, that balsam fir exposed to atmospheric fluoride concentrations averaging 6.3 ppb accumulated 165 ppm fluoride before injury, while low exposures of 0.52 ppb caused injury to the same species at 14 ppm fluoride in tissue. Similar responses were noted in several other plant species. Sidhu concluded that the threshold levels of fluoride that cause plant injury were different among species and also within species at different air concentrations.

There is a consensus among most researchers that sensitive plant species will accumulate less fluoride than more resistant species exposed under similar conditions, no matter what the exposure level. This results from changes in the biological functions of sensitive plants, reducing stomatal efficiency and the uptake of airborne fluoride. Attempts to classify the susceptibility of plant species by fluoride accumulation have had limited success (Weinstein, 1977; NAS, 1971; Threshow and Pack, 1970).
As shown by Sidhu (1978), it is possible to relate plant sensitivity to

fluoride accumulation but only when exposure conditions are the same for each species.

Dose-Response Effects

Numerous plant species are sensitive to airborne fluoride (primarily hydrogen fluoride). Experiments both in the field and in the laboratory have been performed to define plant response at various fluoride levels and exposure durations. Table III.G-II indicates a number of these experiments.

It is clear from Table III.G-II that responses to airborne fluoride occur at several levels of functioning in the plant. Hydrogen fluoride adversely affects photosynthesis in barley and alfalfa plants at 5.0 ppb for two hours (Bennett and Hill, 1973). Increased respiration in bush beans results from exposure to 1.8 ppb of fluoride for 24 hours (Applegate and Adams, 1960).

Hydrogen fluoride exposure to vegetation also has been shown to cause a loss in reproductive capacity and yield. Facteau _et al_. (1973) found decreases in pollen germination and pollen tube growth when sweet cherry, in its flowering stage, was exposed to fluoride. The severity of these effects were directly related to the concentration of hydrogen fluoride with little to no pollen tube growth found at an exposure of 3.1 ppb for 24 hours. Pack and Sulzback (1976) and Pack (1972) found that 0.64 and 0.55 ppb hydrogen fluoride reduced the number and size of fruit in soybean and strawberry respectively, with longer exposures.

Conifers and gladiolus are extremely sensitive to visible injury from airborne fluoride. Gladiolus plants are so sensitive that they are often used as biological indicators of fluoride pollution (NAS, 1971). Table III.G-II shows that the youngest rapidly growing needles of ponderosa pine are injured at concentrations near 1.0 ppb for 24 to 48 hour exposures (Adams _et al_., 1956a).

TABLE III.G-II SUMMARY: FLUORIDE EFFECTS TO SELECTED MONTANA PLANTS

Plant Species	Concentration	Exposure	Response of Vegetation	References
Barley & Alfalfa	5.0 ppb	2 hours	Decreased photosynthesis	Bennett & Hill, 1973
Bush Beans	1.4-5.0 ppb	24 hours	Increased respiration	Applegate & Adams, 1960
Soybean	0.64 ppb	2352 hours (98 days)	Late flowering, smaller paler flowers, reduced fruit and seeds per plant, (fresh weight)	Pack and Sulzbach, 1976
Sweet Cherry	3.1 ppb	24 hours	Little to no pollen tube growth indicating loss of fruit production	Facteau _et_ _al_. 1973
Strawberry	0.55 ppb	2352 hours (98 days)	Reductions in number of seeds and weight and size of fruit (fresh wt.)	Pack, 1972
Gladiolus	0.10 ppb	840 hours (35 days)	Visible injury	Thomas, 1956
Ponderosa pine	0.50 ppb	224 hours (9 days)	Visible injury to 100% of the ponderosa pine exposed in laboratory	Solberg _et_ _al_. 1955
	1.5 ppb	48 hours	Visible injury to 80% of the ponderosa pine exposed in laboratory	
	5.0 ppb	16 hours	Visible injury to 80% of the ponderosa pine exposed in laboratory	
Ponderosa pine	1.22 ppb	24 hours	0.5 Injury Index,[2] young expanding needles	Adams _et_ _al_. 1956a
Ponderosa pine	1.22 ppb	48 hours	1.0 Injury Index,[2] young expanding needles	
Ponderosa pine	0.49 ppb	360 hours (15 days)	6.3% Injury Index[1] in current years needles near Mead, Washington Aluminum Smelter	Adams _et_ _al_. 1956b
	0.77	360 hours (15 days)	18.2% Injury Index[1] in current years needles near Mead, Washington Aluminum Smelter	
Balsam fir	0.55 ppb	744 hours (31 days)	5.0 Damage Index,[3] tissue fluoride= 9.24 ppm. 10.3 km from phosphorous plant	Sidhu, 1977
Balsam fir	0.24 ppb	3600 hours (150 days)	Initiation of tissue damage tissue fluoride = 14.0 greater than 12.0 km from phosphorous plant	Sidhu, 1978
Eastern larch	0.48 ppb	624 hours (26 days)	10.0 Damage Index,[3] tissue fluoride= 17.56 ppm. 10.3 km from phosphorous plant	Sidhu, 1977
White birch, Alder	0.50 ppb	480 hours (20 days)	10.0 Damage Index,[3] tissue fluoride= 11.86 (White birch), 30.99 (Alder). 12.0 km from phosphorous plant.	
Mixed conifer ecosystem	0.13-0.16 ppb	3600 hours (150 days)	15% total plots with visible injury[4] to combined herbs, shrubs, conifers Plot fluoride = 16 ppm Area affected = 25,000 acres	Carlson & Dewey, 1971
Mixed conifer ecosystem	0.16-0.21 ppb	3600 hours (150 days)	46% total plots with visible injury[4] to combined herbs, shrubs, conifers. Plot fluoride = 34 ppm Area affected = 34,000 acres	

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[1] Injury Index = the average needle length divided by the average length of injured tissue then multiplied by the percentage of needles injured.

[2] Injury Index = the percentage of total needles with tissue death multiplied by the ratio of the average length of injured tissue to the average needle length of the needles measured.

[3] Damage Index = the percentage of needles or leaves with fluoride symptoms multiplied by the ratio of length (needles) or distance from margin to midrib (broad leaves) showing damage. The damage index represented the summation of three independent measurements of the tree.

[4] Visible Injury = all vegetation at plot with the percentage of needles or leaves showing fluoride symptoms multiplied by the ratio of the length of injury equalling 0.006 or higher.

The evidence put forth previously in this section indicates that injury to pine needles can cause growth losses and eventual death in affected trees with continued exposure.

Sensitive conifers and other plants sharing their coniferous ecosystem show injury when exposed for a month to hydrogen fluoride levels between 0.25 and 0.50 ppb (Sidhu, 1977 and 1978). Levels between 0.13 and 0.21 ppb may damage these plants when exposed for the length of a growing season (Carlson and Dewey, 1971). Carlson and Dewey (1971) and Sidhu (1977 and 1978) show the cumulative nature of fluoride uptake in plants and the large areas affected by fluoride pollution around emission sources.

Pollutant Interactions

Air pollutants, rarely, if ever occur alone under normal ambient conditions. Some plant species respond differently to pollutant combinations than to single pollutants. The effects from pollutant combinations may be additive, antagonistic, (tending to cancel each other) or synergistic in generating plant response (Reinert et al. 1976). Since hydrogen fluoride and sulfur dioxide are emitted simultaneously from many industries, experiments have been conducted on the combined effects of these pollutants on vegetation.

Matsushima and Brewer (1972) studied the influence of hydrogen fluoride and sulfur dioxide on citrus. High concentrations of 2.3 to 17.1 ppb hydrogen fluoride and 0.8 ppm sulfur dioxide for 23 days caused reductions in the growth length of branches and leaf area. The reductions were greater than when similar plants were exposed to single pollutants. Mandl et al. (1975) found that a seven day exposure to a mixture of 0.15 ppm sulfur dioxide and 0.60 ppb hydrogen fluoride caused about the same amount of visible injury to pinto beans and corn as sulfur dioxide administered alone. Hydrogen fluoride alone caused no visible injury.

At a lower sulfur dioxide concentration (0.6 ppm) and similar hydrogen fluoride levels,a 21-day exposure caused synergistic visible injury to pinto beans and corn.

Particulate Fluoride

Particulate fluoride is less injurious to vegetation than gaseous fluoride compounds. Pack et al. (1959) reported that 15 percent of gladiolus leaf surface was killed when plants were exposed for four weeks to 0.96 ppb hydrogen fluoride. No leaf tissue was killed by exposure to 1.9 ug/m^3 fine particulate fluoride over the same time period. Leaves were analyzed for fluoride after both experiments and showed approximately 33 percent less fluoride content from particulate matter than from gaseous forms. McCune et al (1965) reported less injury to gladiolus and less fluoride accumulation in washed leaves from cryolite (sodium aluminum fluoride dust) than from hydrogen fluoride. Both investigators attributed the low phytotoxicity of particulate fluoride to its inability to penetrate into the internal tissue of the leaf.

Effects on Livestock and Other Animals

Livestock

The harmful effects of elevated fluoride levels on the health of livestock have been well documented (Greenwood et al. 1964; Suttie, 1977; NAS, 1971). Aschbaker (1973) wrote that "of all airborne pollutants which may affect farm animals, fluorine has caused the most serious and widespread damage." Livestock is most likely to be exposed to harmful fluoride levels through the consumption of contaminated forage. A long-term effect (fluorosis) arises when cattle eat vegetation containing more fluoride than normal over an extended period. The symptoms of fluorosis include abnormalities in the teeth, bone and biological functioning (NAS, 1971). Excessive fluoride consumption by cattle during formation of the teeth (up to age four in cattle) causes tooth discoloration and rapid wear. Consumption of contaminated forage over a substantial period leads to fluoride intake above "normal" dietary levels and can cause severe bone abnormalities and lameness. The biological effects of excessive fluoride consumption are a loss of appetite, decreased weight gain and diminished milk yield. Livestock damage from fluorides in Montana has been reported around Ramsay (Miles _et al_. 1978), Garrison (Prival and Fisher, 1973), and Columbia Falls (EPA, 1974).

The potential harmful effects on livestock from high fluoride in forage has prompted Montana and other states to set standards limiting fluoride in forage. The current Montana rule is 35 ppm fluoride in forage. Suttie (1969b) wrote that the only practical protection from livestock fluorosis appears to be a standard based on forage fluoride concentration. The concept of using forage fluoride content as the basis of a standard to protect livestock is accepted by the scientific community studying fluoride effects, but the safe level for fluoride in forage is not absolutely certain. Suttie (1969b) advocated setting a yearly fluoride average of 40 ppm, with allowances for increases to 60 ppm for two consecutive months or 80 ppm for one month. Suttie has conducted various controlled experiments where animals have been fed fluoride compounds and the various responses to fluorides have been identified (Suttie _et al_. 1957; Suttie _et al_. 1961). Suttie (1977) recognized fluoride toxicity symptoms occurred in teeth and bones of animals consuming forage with less than 40 ppm fluoride. He did not, however, conclude that these symptoms were sufficient to influence the over-all health, well-being or economic value of the animals. Greenwood _et al_ (1964) conducted long-term controlled experiments with dairy cows to determine the influence of dietary fluoride on health, meat and milk production. The cows were fed

12, 27, 49, and 93 ppm fluoride (dry weight feed basis) from age three to four months until they were killed at 7.5 years of age.

The research showed that:

1. Milk production is affected if cattle are fed lifetime average concentrations of over 49 ppm fluoride on a total dry diet basis.
2. No milk production effects were observed in animals fed 27 ppm fluoride on a total dry weight diet.
3. Cattle fed diets of 27 ppm fluoride had slightly mottled incisor teeth and very slight bone fluorosis but no impairment of body function.
4. Moderate to marked dental effects and slight to moderate bone fluorosis occurred in cows fed 49 ppm fluoride.
5. Lameness and excessive bone and tooth effects were observed in cows fed 93 ppm fluoride.

Elevated levels of bone fluoride are the best single measurement of fluorosis in cattle (Suttie et al. 1961). These experimenters fed controlled levels of fluoride to cattle over varying periods and found that resultant bone fluoride levels of 4000 to 5000 ppm (dry fat-free weight) were associated with excessive tooth staining and wear. The researchers slaughtered the cattle and noted thick, rough textured abnormalities in the bone.

When considering the influence of fluoride on the health of livestock, it is important to recognize that most of the research has been conducted under laboratory conditions. The National Academy of Sciences (1971) cautioned that care must be taken in applying the results of laboratory experiments to field situations where there may be a highly variable relationship between the cycling of fluoride in the ecosystem and fluoride intake in livestock. Shupe (1978) said his field observations indicate that fluorosis symptoms in cattle become evident at lower concentrations of forage fluoride under natural conditions than in the laboratory.

Miles et al. (1978) conducted a field and literature study of fluoride pollution near a phosphorous plant at Ramsay and the effects on livestock and selected wildlife in the area. There were documented accounts of one ranch where 98 percent of the heifers had damaged teeth in 1977. Nine spot samples of forage from the ranch in October 1977 showed an average fluoride level of 18 ppm. On another ranch in the area with an average fluoride level of 10 ppm in ten forage samples, five to six cows examined had marked to severe tooth damage from fluoride. In addition, two ten-year-old cows had permanent teeth erupting through the gum line indicating severe dental injury.

The Miles et al. (1978) study also suggests that horses may be more sensitive to fluoride than cattle. Three horses sampled for bone fluoride showed that a level of 3200 ppm in the foot bones (metatarsus) was associated with bone and dental abnormalities. Suttie et al. (1961) found that 4000 to 5000 ppm was the threshold for similar symptoms in cattle.

Another field study conducted in Canada agrees with the findings of Miles et al. (Krook and Maylin, 1978). Results from this study indicate that fluoride levels of 2477 to 5753 ppm in cattle vertebrae were associated with growth loss, severe dental fluorosis, lameness, and difficulty chewing and drinking. Over 200 unwashed vegetation samples analyzed for fluoride throughout the growing season ranged in average concentration from 9 to 32 ppm at various sampling sites throughout the study area. An unborn seven-month-old calf fetus had a bone fluoride level of 500 ppm, a higher bone fluoride content than normally found in fully grown cows from uncontaminated areas (Suttie, 1977). This indicates transfer of fluoride between the mother and unborn calf. Krook and Maylin studied the adverse affects of fluoride to developing bone and concluded that unborn or newly born cattle are particularly sensitive to abnormal bone and teeth formation from low concentrations of fluoride when exposure is continual. This sensitivity in young animals indicates that bone fluoride levels are not the best indicator of adverse effects in all cattle. Also, the work of Suttie indicating a threshhold effect of 4000 to 5000 ppm in cattle was based on laboratory studies, which may underestimate by a considerable margin the fluoride levels necessary to cause health damage to animals under long-term field conditions. The results also suggest that cattle fluorosis may occur under field conditions at lower levels of forage fluoride than the levels known to cause fluorosis under laboratory conditions. This view is similar to that held by Shupe (1978).

Wildlife

The influence of airborne fluorides on the population dynamics and health of wildlife is not well understood although there are publications concerning fluoride content and accumulation in bones. Newman and Markey (1976) investigated the effects of dietary and bone fluoride on the health of deer mice under laboratory conditions. At dietary fluoride concentrations above 1300 ppm and resultant upper leg bone fluoride levels of approximately 3000 ppm, the experimenters noted weight loss, increased death rates, changes in leg bone size, and tooth malformation after eight weeks. Tourangeau et al. (1977) measured the upper leg bone fluoride content in deer mice around two coal-fired generating plants before and after the plants began operating. It was speculated the increases in bone fluoride from 200 to 600 ppm might have resulted from the generating plants' fluoride emissions or from normal seasonal variations in fluoride uptake. Miles et al. (1978) measured jawbone fluoride content of rabbits in the Ramsay area and reported levels ranging from 689 to 5657 ppm, or 2 to 35 times as high as in rabbits from uncontaminated areas.

Natural bone fluoride levels in seven species of hoofed wild animals found in Montana have been studied (Kay et al. 1976). Fluoride was found to increase with the age of the animal and to vary from place to place. In general, jawbone fluoride in elk and mule deer from uncontaminated areas averaged 113 ppm and 83 ppm, dry fat-free weight. Karstad (1967) reported that whitetail deer collected in the vicinity of an industrial complex had jawbone fluoride levels of 4300 to 7125 ppm, with associated pitting and discoloration of teeth, abnormal tooth wear, and fractures of the teeth and jaw bones. Bone abnormalities were observed at jawbone fluoride levels as low as 2200 ppm.

Elevated bone fluoride has been found in big game animals from two locations in the state. Miles et al. (1978) measured fluoride in the jawbone of one elk and one mule deer near Ramsay. Results showed a fluoride level of 1771 ppm in the elk and 1619 ppm in the mule deer. These levels are 17 times the amount found in specimens from uncontaminated areas of the state. Kay et al.(1975b) found that jawbones from two mule deer collected from Teakettle Mountain near Columbia Falls contained 6300 to 3180 ppm fluoride. Jaws from two whitetails from the same area contained 9760 to 6700 ppm fluoride. The authors hypothesized that lameness noted in the deer was the result of excessive exposure to fluoride and subsequent accumulation in their bones.

Insects

Bees and other insects are important as pollinators of agricultural and native plants in Montana. In addition, bees often are raised commercially for wax and honey. Montana ranks among the top states for total production from bee colonies. Approximately 25 percent of Montana's 80,000 hives are taken temporarily to other states each year to pollinate agricultural crops (Bromenshenk, 1978).

Pollinating insects are susceptible to fluoride pollution in two ways: direct inhalation, and ingestion of contaminated nectar and pollen. Laboratory experiments with honeybees indicate that continuous exposure to fluorine gas at four to five ppb for up to eight weeks caused a 13 percent recution in the lifespan of worker bees. This reduction occurs during the period when workers should be engaged in pollinating and foraging activities - both important functions of a healthy bee colony (Atkins, 1970).

Pollinating insects in contaminated areas tend to accumulate fluoride in their bodies through their foraging behavior. Lillie (1970) said a fluoride level above 30 ppm in honeybees always indicates the presence of a fluoride emission source. Bromenshenk and Gordon (1978) reported elevated fluoride levels in honeybees near fluoride emitting sources in Montana. Carlson and Dewey (1971) found the fluoride content of honeybees, bumble bees, and sphinx moths near Columbia Falls to be 221, 406, and 394 ppm respectively. These levels were several hundred times higher than fluoride found in insects from uncontaminated parts of the state.

Several studies have identified a level of fluoride accumulation toxic to pollinating insects. Bromenshenk (1978) studied fluoride accumulation in honeybees from throughout Montana and concluded that 120 to 170 ppm (dry weight) is toxic to 50 percent of the population. Similarly, Lillie (1970) reported bees died from an accumulation of 130 to 150 ppm fluoride in their bodies.

Another aspect of insect/fluoride relationships is the influence of air pollutants on populations of insect pests. Numerous studies have documented that air pollutants predispose trees to infection by insects (Stark, et al., 1968; Stark and Cobb, 1969).

In Montana, Carlson et al. (1974) studied the relationship between an insect infestation of lodgepole pine and levels of fluoride emitted from the nearby Anaconda Aluminum Smelter. They found that needle miner and pine needle sheath miner epidemics near the facility caused damage increments that could be statistically related to airborne fluoride levels. It was determined that excessive fluoride exposure decreased needle resistance to insect attack causing a buildup of parasitic insects, resulting in tree damage.

Effect on Ecosystems

The effects of fluorides on ecosystems result from its persistence and accumulation in the environment. In Montana, studies conducted near the Anaconda Aluminum Smelter at Columbia Falls by the EPA (1974), Carlson and Dewey (1971), and Dewey (1973), near the Stauffer Chemical Co. at Ramsay (Miles et al. 1978), and surrounding Rocky Mountain Phosphate Co. plant at Garrison (Kay, 1974), indicate significant accumulations of fluoride in vegetation, insects, and plant-eating mammals. The potential for fluoride accumulation in humans from contaminated foodstuffs and drinking water has also been documented (Rippel et al.,1976). The effects of fluoride in ecosystems are shown in several studies (Carlson and Dewey, 1971), (Carlson, 1974), (Dewey, 1973) on uptake of fluoride by insects and the relationship between insect pests and fluoride increases in trees. These studies suggest three important consequences of fluoride effects within ecosystems:

1. Fluoride concentrations are magnified through some elements of the food chain, with a given fluoride level in vegetation leading to a higher concentration in insects consuming the plant, and a still higher concentration in predatory insects.

2. A relationship between insect infestations and fluoride levels in conifers indicates fluoride accumulation in trees may make them susceptible to insect pests.

3. Among insect studies, pollinating species are the most sensitive to fluoride. This could result in alterations to plant communities, since many plant species depend on these insects for pollination.

Plant community structure has been shown to be directly affected by a number of air pollutants; e.g., ozone, (EPA, 1977) and sulfur dioxide, (Gorham and Gordon, 1960). Williams (1978), in a study of fluoride damage to a Douglas fir forest near Columbia Falls, Montana, showed that fluoride pollution changes the plant community structure of forests, shifting it away from tree species to a less aesthetic and economically important shrub-grassland plant community. Williams said in her study that attempts by forest managers to reseed trees in fluoride-impacted areas would prove futile without curtailment of fluoride pollution.

Lower plant species such as lichens and bryophytes had been shown to be essential to the normal functioning of agricultural and natural ecosystems (see pp 91). LeBlanc _et_ _al_. (1972) has shown that fluoride pollution reduces the number of lichens and bryophyte species near a fluoride emission source. Even when some specimens were able to maintain themselves close to a source, the researchers found that representatives of the same species were five times more plentiful in areas without elevated fluoride levels.

Effects on Materials

High concentrations of hydrogen fluoride etch glass (NAS, 1971). No other material effects are known to result from airborne fluorides.

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III.H. HYDROGEN SULFIDE

Introduction

Hydrogen sulfide is a highly odorous gas with a rotten egg smell. It is produced by some industrial processes and the natural decay of plant material. The principal sources of hydrogen sulfide in Montana are the Hoerner-Waldorf pulp mill, oil refineries and oil and gas production fields. Additional information on the sources and ambient measurements of hydrogen sulfide can be found in Sections II.B and Appendix A.

Hydrogen sulfide at concentrations measured in Montana have been shown to adversely affect human health. Hydrogen sulfide can produce nausea and shortness of breath. Persons with psychological problems of paranoia or schizophrenia may be especially sensitive to hydrogen sulfide.

Adverse effects on crops of the types grown in Montana have been observed.

Human Health Effects

The odor of hydrogen sulfide is perceivable to most people at concentrtions between 0.0015 and 0.0075 ppm (Wilby, 1969). Hydrogen sulfide quickly fatigues odor sensors in the nose, so residents of an area with constant exposure to hydrogen sulfide will not notice an odor at the usually perceivable concentrations. It is not clear how long the odor level must be reduced for the nose to recover its sensitivity. If the concentration significantly increases the odor can easily be perceived (Public Health Service, 1964a).

A ten-year study of eye damage (conjunctivitis) among workers exposed to both carbon disulfide and hydrogen sulfide indicated a threshold for injury of about 20 ppm (Barthelemy, 1939). At higher levels, pneumonia and irritation of the throat and lungs have been observed (McCabe and Clayton, 1952).

Community exposures to high levels of hydrogen sulfide in Terre Haute, Indiana and Alton, Illinois led to a substantial number of complaints of shortness of breath, headache and nausea (Illinois Institute for Environmental Quality, 1974). The concentrations of hydrogen sulfide were approximately 0.3 ppm in Terre Haute (Public Health Service, 1964b).

Effects of small hydrogen sulfide doses on alcoholics or others who have consumed substantial amounts of alcohol may result from psychological reactions related to the odor of the gas (Poda, 1966). The condition of neurotics and persons with paranoid or schizoid tendencies appears to be much worse after hydrogen sulfide exposure (Poda, 1966).

Effects on Crops and Other Plants

Hydrogen sulfide slows plant growth. It has been shown to decrease photosynthesis (Knobloch, 1966) and to inhibit other important functions in the plant (Day and Franklin, 1946).

In the needles of conifers, hydrogen sulfide causes tissue death, distinct from similar effects caused by weather, fungus and insects, or soil variations. The damage caused by hydrogen sulfide appears the same as that caused by either sulfur dioxide or methyl mercapatans, implying that the toxicity mechanisms are similar (Carlson, 1978).

Relatively few dose-response studies have been performed to determine the effects of hydrogen sulfide on plants, largely because experiments done over 30 years ago seemed to indicate that even heavy concentrations (up to 20 ppm) over a period of hours did not cause damage even in sensitive plant (McCallan and Wilcoxon, 1931; McCallan *et al*., 1936; Setterstrom and Thornton, 1940). Recent studies using modern methodology have reversed the earlier conclusions and shown hydrogen sulfide to be harmful to vegetation below one ppm over prolonged exposure periods. (Thompson and Kats, 1978; Bennett *et al*., 1978)

TABLE III.H-I

Vegetation Effects from Hydrogen Sulfide (Thompson and Kats, 1978)

PLANT SPECIES	CONCENTRATION	EXPOSURE	RESPONSE OF VEGETATION
Alfalfa (El Dorado)	0.03 ppm	28 days	Cutting #1 2% Dry wt. loss
		35 days	Cutting #2 2% Dry wt. loss
	0.30 ppm	28 days	Cutting #1 19% Dry wt. loss
		35 days	Cutting #2 31% Dry wt. loss
Alflafa (Hayden)	0.03 ppm	28 days	Cutting #1 0% Dry wt. loss
		35 days	Cutting #2 7% Dry wt. loss
	0.30 ppm	28 days	Cutting #1 38% Dry wt. loss
		35 days	Cutting #2 39% Dry wt. loss
Sugar Beets	0.03 ppm	123 days	5% sugar content loss in root 27% Fresh wt. gain in root
	0.10 ppm	123 days	6% sugar content loss in root 25% Fresh wt. loss in root
	0.30 ppm	123 days	18% sugar content loss in root
Ponderosa pine	0.03 ppm	76 days	elevated sulfur in foliage, tip burn after 56 days
	0.30 ppm	76 days	loss 4 year needles, decrease in 3 year needles, tip burn after 70 days
Douglas fir	0.03 ppm	246 days	no tip burn or needle color change
	0.10 ppm	246 days	tip burn in needles, reduction in growth and dry weight
	0.30 ppm	246 days	tip burn in needles, reduction in growth and dry weight

Thompson and Kats (1978) studied growth/yield and leaf/needle effects of hydrogen sulfide on alfalfa, sugar beets, ponderosa, and Douglas fir over an extended exposure period (Table III.H-I). The threshold for significant injury in these plants was between 0.03 ppm and 0.3 ppm, except Douglas fir, which had a threshold between 0.03 ppm and 0.1 ppm for a 246 day exposure. All species examined in this study showed increased sulfur accumulation following exposure to hydrogen sulfide. The sulfur levels measured in the plants corresponded to the exposure levels. Although some plants were more sensitive than others to hydrogen sulfide, the degree of sensitivity did not relate to the rate of sulfur accumulation. Fast growing plants generally accumulate sulfur more rapidly than slow growing plants.

The Thompson and Kats (1978) experiments with sugar beets indicated that exposure to hydrogen sulfide concentrations above 0.03 ppm caused an increase in the weight of sugar beet roots and a reduction in sugar content, thus reducing the market value. The effects on sugar beets suggest hydrogen sulfide creates a carbohydrate deficiency in the roots similar to that observed in other root crops exposed to various air pollutants (Tingey et al., 1971).

Thompson and Kats (1978) also observed that the yields of Hayden and El Dorado alfalfa varieties were not reduced at 0.03 ppm hydrogen sulfide but that 0.3 ppm reduced growth significantly. The accumulation of sulfur as sulfate in the leaves corresponded to the concentrations in the air, ranging from an average of 1330 ppm in the leaves at an exposure at 0.03 ppm to a mean 2930 ppm at 0.3 ppm for 2 harvests (Thompson and Kats, 1978).

With ponderosa pine, a ten-week exposure to 0.03 ppm hydrogen sulfide had no visible effect. Sulfate accumulation in the plants was proportional to the level of the hydrogen sulfide exposure. At 0.3 ppm there was a significantly greater drop of three year old needles, and the fourth needle whorl was completely defoliated. Such defoliation in ponderosa pine interferes with plant growth because the older needles act as a temporary repository for nutrients that move to growing tissue when demand occurs (Kozlowski, 1973).

Bennett et al. (1978) did field experiments with snap beans grown under field conditions. They found significant visible leaf injury after 55 days of four-hour daily hydrogen sulfide exposures both alone and in combination with ozone. The bean plants were visibly injured by hydrogen sulfide both with and without ozone at concentrations above 2.0 ppm. Below 2.0 ppm there was no visible injury at relatively short 95 to 100 hour exposures up to the time of pod set.

These investigations determined that a combination of hydrogen sulfide and ozone had a significantly greater effect than hydrogen sulfide alone in reducing the total yield, measured as dry weight of the pods and the whole plants. Bennett et al. suggested that hydrogen sulfide acting alone sets off a different toxicity mechanism than when it is combined with ozone. They postulated a significantly more toxic interaction occurred when the two are mixed. The researchers concluded that hydrogen sulfide doses greater than 400 ppm-hours (4.0 ppm x 100 hours) in the absence of ozone and approximately 100 ppm-hours in the presence of ozone should be avoided to prevent serious losses in bean crops.

Assuming that the dose-response function of hydrogen sulfide is constant over a large range of concentrations and exposure periods, the injurious dosage suggested by Bennett et al. (1978) translates to 0.40 ppm x 1000 hours (about six weeks) without ozone or 0.1 ppm x 1000 hours with ozone. This approximately corresponds to the sensitivity of alfalfa as determined by Thompson and Kats.

The adverse effects of hydrogen sulfide on plants under natural conditions have been studied by several investigators. Thomas et al. (1950) and McAllister (1976) found damage to lodgepole pine near geysers and hot springs in Yellowstone National Park. Both researchers noted that damage and sulfur levels in the vegetation was highest in plants nearest the sources and declined with distance from thermal areas. McAllister identified hydrogen sulfide as the cause of tree damage. An average concentration of 0.085 ppm was associated with the heaviest damage noted and 0.05 ppm was present in areas with moderate damage. These researchers suggested that lower levels of hydrogen sulfide can cause damage under natural conditions than under controlled exposure conditions. Naturally growing plants in the field are subject to stresses other than pollution, such as weather and soil conditions, which may make them more susceptible to pollution damage than laboratory grown plants.

Carlson (1974) suggested that sulfur damage to Douglas fir near the Hoerner-Waldorf polp and paper mill in the Missoula Valley was caused in part by hydrogen sulfide. He suggested that unseasonable weather conditions and sulfur dioxide emitted from the mill also may have contributed to the damage. Carlson's study found substantially more injury to Douglas fir than ponderosa pine, conflicting with the findings of Thompson and Kats (1978).

Effects on Livestock and Other Animals

Exposure to high concentrations in Poza Rico, Mexico caused livestock losses of 50 percent (McCabe and Clayton, 1952).

Effects on Ecosystems

No effects have been recorded.

Effects on Materials

Hydrogen sulfide can darken lead-based paints and tarnish metals. Lead-based paints are most easily affected by hydrogen sulfide with blackening caused by concentrations of 0.05 ppm over a two-hour period. Metals such as silver and copper tarnish rapidly in the presence of 0.0027 ppm hydrogen sulfide over 50 hours (Wohlers and Feldstein, 1966).

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IV. ALTERNATIVES AVAILABLE TO THE DEPARTMENT

IV.A. INTRODUCTION

Following the decision that the state air quality standards might not be enforceable, the Board of Health and Environmental Sciences indicated it wanted the Air Quality Bureau to develop standards that could be enforced. The question of what pollutants should be controlled by the standards, and how the standards should be developed and enforced must be resolved by still unmade policy decisions.

Although there are existing Montana ambient air quality rules for suspended sulfates, reactive sulfur (sulfation), sulfuric acid mist and beryllium, the Air Quality Bureau staff does not believe that sufficient data are available to propose new standards for these. The bureau will continue to review the scientific information on the effects and measurement techniques for these pollutants, with the intent of proposing standards when adequate data are avilable. The bureau also is reviewing the scientific information available on cadmium and arsenic, and polycyclic organic material with the intent of eventually proposing standards for these. It was further decided that a standard should be considered for visibility, as required in specific areas by the 1977 amendments of the Federal Clean Air Act.

When pollution data was reviewed for its applicability to the Montana pollution situation, it became clear that four standards originally adopted should be dropped, at least temporarily. The beryllium standard can be dropped because there is no source of beryllium in Montana and no apparent

prospect that there might be such a source. In addition, beryllium is covered by the National Emissions Standards for Hazardous Air Pollutant Sources rule which has been adopted by the Board of Health and Environmental Sciences. The rules for suspended sulfates and sulfuric acid mist should be dropped because the available data is not sufficient to define what standards should be set. A standard for sulfuric acid mist would be impractical because of the difficulty of operating ambient measuring techniques accurately under field conditions. A respirable particulate standard was considered, but shelved until adequate research is performed.

Total reactive sulfur was dropped from the list of regulated pollutants because of the ambiguous results yielded by the sulfation plate measurement method.

There was some consideration of setting standards for cadmium and arsenic, both of which are emitted in substantial quantities by Montana sources but neither of which has been controlled under a state ambient air standard in the past. It was decided that currently available research results do not define a safe concentration level for these two pollutants, so no standards were considered for recommendation. Such standards will be considered when the data is sufficient to determine what concentrations should be allowed. Summaries of the data concerning pollutants for which no standards are proposed are found in Appendix F.

Research indicated there was sufficient data to allow recommendation of standards for sulfur dioxide, carbon monoxide, photochemical oxidants, nitrogen dioxide, lead, fluoride and particulates, including both total suspended particulates and settled particulates. Once it was determined what standards could be scientifically and technically justified, there remained a variety of alternatives to be considered.

IV.B. PROPOSE STANDARDS EQUAL TO THE FEDERAL STANDARDS

The board made clear that the bureau was to research and recommend legally enforceable ambient standards. Federal law requires the bureau to consider state standards at least as stringent as the federal standards. If state standards were set less stringent than the federal primary standard, pollution sources still would have to comply with the federal standard. Setting a state standard more lax than a federal secondary standard would only delay for a few years the stricter emission controls that would be required before some sources have to meet the federal secondary standard.

Furthermore, most of the federal standards are several years old, and are based on the now outdated information that was available when they were set. Like the former state standards, many of the current federal standards need review and updating. The EPA is performing some review, but it will not be complete for all the pollutants for several years. It was precisely the purpose of the MAAQS Study to review information published more recently than that upon which the older federal standards are based.

The suitability of the federal standards for Montana is limited, because the pollution situation here is different than in much of the rest of the nation. The Federal standards were designed as a compromise to regulate pollution under a wide variety of circumstances, for example, in heavily industrialized large cities. Under such conditions, it often is not possible to determine which of many sources is responsible for ambient pollution violations. In Montana, by contrast, major industrial sources tend to not be located near other sources, which often makes it much easier to determine what facility is causing ambient violations.

The presence in Montana of two major national parks, numerous wilderness areas and seven Indian reservations, all of which are prized for their relatively pristine environmental conditions, further separates Montana from the pollution conditions envisioned when the older federal standards were set. It is clear that Montana needs ambient air standards which reflect the actual conditions in the state and the desires of the people, as expressed in the state laws and constitution. For all the above reasons, the limits set by federal standards, although a useful reference, were not specifically considered for adoption as state standards. The scientific data was reviewed with the specific purpose of determining what standards would protect the health of Montanans and to guard the quality of the environment and economy of the state from the effects of air pollution.

IV..C. STANDARDS STRICTER THAN FEDERAL STANDARDS

There is a wide range of theoretical standards that could be considered but most of these would be only of academic interest. The possible standards requiring serious consideration are those which would provide an adequate margin of safety to protect health and the environment, and which can be met in large part without unnecessary economic strain on the owners of pollution sources or the public. To determine what pollution levels would be defined by such standards, it is necessary to perform detailed technical review of the various characteristics of pollutant streams within sources. Only when these details are known can the cost of reducing emissions by various degrees be estimated. Often, however, such estimates are limited in their accuracy by the unavailability of some of the pertinent information, particularly the costs of necessary equipment.

Furthermore, most information concerning the ability or willingness of an industry to finance pollution controls is known only to the company, and not available for evaluation by others. Other variables, such as the market price of the product, the age, condition and profitability of a given plant, and the energy demands of a given control, all are unknown to varying degrees, and further complicate the process of calculating what standards can be met.

Despite pitfalls, MAAQS reviewed all pertinent and reasonably available information to enable the Air Quality Bureau to evaluate the ramifications the setting of certain standards would have on the economics and operations of some industrial facilities. Although the costs attached to the various control options are of interest, cost is not considered in determining what standards are necessary to protect health.

Economic Considerations in Proposing Standards

ASARCO

ASARCO recently installed a new sulfuric acid plant to reduce sulfur dioxide emissions from a portion of the ore sintering machine. Also, the company has altered the sinter machine so that most of the sulfur dioxide it generates is recirculated until it becomes concentrated enough to be treated in the acid plant. All these new measures together result in a total sulfur dioxide control level of about 75 percent. To further reduce ground level sulfur dioxide concentrations, the plant managers have agreed to install a new 375 foot stack to elevate blast furnace emissions and reduce ground level concentrations.

Computer simulation of atmospheric conditions in the East Helena vicinity indicates that the new controls will reduce ambient concentrations enough to comply with the federal ambient standards. Whether such compliance actually will be achieved will not be known until very stable weather conditions trap the emissions in the valley and cause ambient concentrations to reach the maximum.

If further controls were required to meet ambient standards, a currently uncontrolled sulfur dioxide stream off the sinter machine could be recirculated to increase its concentration and make it treatable in the acid plant. This would increase total sulfur dioxide control to approximately 80 percent. Such alteration probably would not require any substantial modifications of the sinter machine. Cost of the modification would be less than $500,000 with an additional annual cost of approximately $100,000. The modification would use a relatively small amount of electrical energy to power fans.

Lead particulate also is released from the plant. Sources relatively close to the ground have more impact on the immediate community than those high in the air, such as stacks. These so-called "fugitive emissions" are primarily dust stirred up by the handling of materials, such as ores, sinter, and slag, and dust escaping from the dross-reverberatory furnace building.

Lead particulate from materials handling can be partly controlled by "good housekeeping" practices, such as sprinkling the fresh slag piles to keep them damp and less likely to be picked up by the wind.

Emissions from the dross-reverberatory building can be controlled by exhausting all the air in the building through a baghouse. This equipment could be installed for about $7 million.

Controls at this level would not, however, eliminate a serious occupational health situation in the building. It would require an estimated $9 million to eliminate both the emission problem and the occupational health problem. The annual operating costs of the $9 million option would be about $1.5 million. This expense would be defrayed to an unknown degree by the value of the lead particles recovered. Energy consumption would be "moderate to moderately high."

Anaconda Copper

A significant portion of the gas stream from the main smelter facility is treated in a sulfuric acid plant, which extracts the sulfur dioxide and converts it to sulfuric acid. This treatment currently results in removal of approximately 34 percent of the total sulfur generated in the process. The current Montana emission rule requires the company to remove 90 percent of the input sulfur. The Environmental Protection Agency (1977) concluded that removal of 86 percent of the input sulfur would bring the smelter into compliance with the federal 24-hour ambient primary standard. Compliance with the current state 24-hour emission rule would require removal of 90 percent. Computer modeling of atmospheric conditions in the Anaconda vicinity indicates that 86 percent control would result in ambient sulfur dioxide concentrations below 0.02 ppm, annual average.

Control of 86 percent of the input sulfur would require additional acid plant facilities equal to about 60 percent of the current 990 ton per day acid production capacity. The cost of the new plant and the necessary duct work and stack should be approximately $21 million. The net capital and operating costs of such facilities (in today's dollar values) over a 30-year period is estimated at $48.5 million. The market for sulfuric acid is limited in the Northwest, which could require neutralization and disposal of additional acid produced at Anaconda. If neutralization were necessary, the total operating costs of the suggested sulfur dioxide controls are estimated to be approximately $2 million a year. The increased energy demands would be small.

The possibility of additional capital expenditures for any industrial facility always raises the question whether the costs can be sustained by the company. In the case of Anaconda, the copper profit margin is currently so low that additional capital and operating costs diminish the financial prospects of the company. However, in view of the recent new investment in the Anaconda smelter, additional investment to control emissions probably would not cause closure of the plant.

Anaconda's copper operations provide five percent of the total employment in Montana. In 1975, the total employment was 4,500 persons, who supported an additional 10,000 secondary jobs in the provision of goods and services. The current employment figures are somewhat lower. If the smelter were to close, the remaining related operations (Butte mining, B.A.& P. Railroad, Great Falls Refining Plant) also would close (Erickson, 1977).

Cenex

The Cenex refinery in Laurel is the third largest emitter of sulfur dioxide in Montana. Although it has the smallest production capacity of the three Billings area refineries, the use of high-sulfur Wyoming crude oil makes Cenex the largest contributor to ambient sulfur dioxide in the Yellowstone Valley.

The company has agreed to implement process modifications that are expected to reduce current sulfur dioxide emissions from the plant by 25 to 30 percent. Construction of new smokestacks and raising some existing stacks is expected to further reduce ground level sulfur dioxide concentrations.

Maximum annual average sulfur dioxide ambient concentrations following the construction and modifications are expected to be less than 0.02 ppm. Process modifications are expected to increase the efficiency of the plant and result in a net saving of energy.

Conoco

Although the Conoco refinery is similar in production capacity to the other two Billings area refineries, it produces only a third as much sulfur dioxide, primarily because it uses low-sulfur Canadian crude oil. Declining imports of Canadian crude in the future could cause the closure of the plant if suitable substitute crude could not be found. The design of this refinery would not permit use of high sulfur crudes without substantial plant modification.

The Conoco refinery does not appear to contribute significantly to the sulfur dioxide levels found in Billings, so additional controls probably would not be required.

Exxon

The Exxon refinery may be a significant contributer to the annual average sulfur dioxide level of 0.0269 ppm measured at the Lockwood School. There is not sufficient meteorological or monitoring information to allow determination of how much Exxon contributes to the elevated concentration.

Several measures could be used to reduce emissions at the Exxon plant if necessary to meet any standard proposed by the Air Quality Bureau. A sulfur dioxide emission reduction of approximately 30 percent could be achieved by installing a lime scrubber on the coker carbon monoxide boiler exhaust stream. Such installation would require a capital investment of $2.7 million and an annual operating cost of approximately $200,000 per year. Energy demands of the scrubber would be moderately high. Other means of sulfur oxide control that might be feasible at Exxon include desulfurization of refinery fuel oil or crude oil.

Montana Power Plant in Billings

There is not enough meteorological or monitoring information to determine whether the Corette plant contributes significantly to sulfur dioxide concentrations found in the Billings area. If additional sulfur dioxide emission controls were necessary, a lime scrubber could be installed to remove sulfur dioxide from the exhaust gas stream. The cost of this type facility on the Colstrip plants indicates the Corette plant could be controlled to about 30 percent for an installation cost of $6.7 million. This would be enough control to eliminate the plant's contribution to elevated ambient levels. The annual operating cost is estimated at $500,000. The energy demand might be up to two percent of the plant's electrical production.

A space shortage at the site might make it necessary to use holding tanks and sludge dewatering facilities to process scrubber residues before they are trucked to settling ponds. A water conditioner also might be needed to treat river water before use in the scrubber. These items could add an additional $1.2 million in capital costs (Bloom, et al., 1978).

Approximately seven percent of the sulfur in the coal currently is removed by crushing the coal and screening out the pyrite impurities. A flotation process could increase the efficiency of this removal to about 15 percent. This process would also remove about five percent of the heating value of the coal. Construction of the additional coal cleaning facilities would cost approximately $3 million. Operating costs would be about $300,000 per year, including the value of the lost heat value from the coal (EPA, 1978).

Montana Sulphur and Chemical Company

This plant receives sulfur-laden natural gas and hydrogen sulfide from the Conoco and Exxon refineries, removes the sulfur and returns the clean gas to be burned for refinery process heat. The hydrogen sulfide is processed into liquid hydrogen sulfide, sulfur and other chemicals. Most refineries, such as the Cenex plant, have their own sulfur plant rather than use the facilities of a separately owned company.

This plant releases one of the most concentrated sulfur dioxide streams in Montana. The company recently agreed to raise its 100 foot stack to 200 feet in an effort to reduce ground level concentrations of sulfur dioxide. The meteorological information for this area is insufficient to allow determination of how much this plant is contributing to ambient concentrations or whether the planned stack will reduce them.

The current sulfur dioxide emissions could be reduced by approximately two-thirds through the use of tail gas scrubbing equipment designed specifically for installations like Montana Sulfur and Chemical. The cost of such equipment would be about $750,000. For approximately twice that price a more sophisticated tail gas cleaning unit could reduce emissions to about one-tenth their present level.

The operating costs of these units would range from approximately $150,000 a year for the cheaper unit to about $850,000 for the more expensive model. Energy demand would be low for the lesser option and high for the greater. Expenses incurred in installing tail-gas scrubbing units could be passed back to the refineries.

Stauffer Chemical

The major sources of fluorides at the Stauffer plant are the furnaces and slag piles. The furnaces are central to the process of extracting elemental phosphorus from phosphate rock. Contents of the furnaces are processed at high temperatures. Pressure develops within the furnaces, and when the contents are tapped, the pressure causes the release of fluorides.

Emissions currently are controlled to a certain extent by four hoods which collect some of the emissions from the furnaces and conduct them to an electrostatic scrub tower.

Research into the effects of ambient fluoride around the Stauffer plant as detailed in Section III.G. indicates a substantial increase in control efficiency would be necessary to reduce ambient concentration sufficient to adequately protect plants and animals. Such control could be provided by additional exhaust and scrubbing of the same design as the current system but with twice the capacity.

Installation cost of such a system would be approximately $2 million. Operating costs are unknown. Energy demand would be moderate.

Anaconda Aluminum

The present control program at Anaconda Aluminum is expected to substantially reduce emissions. If further control were required, scrubbing units could be installed along the roof of the building to catch escaping fluoride. The cost of such a control program is estimated at approximately $25 million. Operating costs and energy demand are unknown.

Hoerner-Waldorf

This plant is one source in the state known to emit hydrogen sulfide. Computer modeling, emission information, and ambient monitoring indicate that the current level of emission would not violate a standard set to protect health.

Particulates-Point Sources

Few point sources in Montana cause ambient particulate levels sufficient to threaten health. Setting a standard sufficient to protect health is not expected to require additional controls by most sources, such as fuel burning or process equipment. However, open mining operations, both strip mines of the type found in eastern Montana and open pit mines of the type found in Butte, produce substantial amounts of particulate, and sometimes cause violations of a standard set to protect human health. Control costs and methods vary greatly among various sources and it is not yet known what it would cost to reduce emissions from any source to the level of any given standard.

Particulate-Area Sources

Most particulates generated by area sources are relatively coarse and do not travel far or have the capacity to penetrate into the deep lung of humans. These particulates are mainly a nuisance.

Any rule proposed to regulate particulates to levels low enough to protect human health would be violated occasionally by emissions from area sources such as unpaved roads or paved roads or streets with substantial dust deposits. Paving unpaved roads is estimated to cost between $30,000 and $35,000 per mile, assuming no realignment or reconstruction is needed on the roadbed. Although paving costs are relatively high, they are more cost-effective than the continuing application of water or dust suppressant oil.

The cost of cleaning dusty paved streets depends on the frequency and type of cleaning necessary.

Automotive Pollutants

Mobile sources, mainly cars and trucks, are the primary cause of ambient concentrations of carbon monoxide, nitrogen dioxide, hydrocarbons and photochemical oxidants. The photochemical oxidants are not emitted from mobile sources, but are created in photochemical reactions among pollutants emitted by cars.

Carbon monoxide emissions are largely eliminated from car exhausts by catalytic converters. Nitrogen dioxide and other precursors of photochemical oxidants could best be controlled by limiting the use and engine size of automobiles. Other possible controls could be various traffic control plans to eliminate traffic concentrations. The costs of these controls vary according to particular situations, and cannot be readily generalized.

Oil refineries also are believed to contribute to excessive levels of the above pollutants. Costs necessary for industry to control these pollutants are not known.

IV.D ECONOMIC ASPECTS OF AIR POLLUTION

Social Costs

The costs of ill-health and environmental damage associated with air pollution can be estimated. Although the results of experiments establish that health and environmental effects will be observed above certain air pollution concentrations, few experiments have attempted to determine what percentage of the population will be affected at a particular concentration. The population affected at a given pollutant concentration can be expressed with an equation called a "dose response function." Only a few of these are known for plants or for people. The functions for ill-health and mortality must be developed from statistical analyses, subject to all the criticisms attached to such studies. The number of especially sensitive people or plants in a given area is never known and is difficult to estimate. Seldom is the actual distribution of the pollution known.

The results of such statistical calculations are no more than initial cost estimates. It is generally accepted that the lack of information leads to substantial underestimates of the actual costs.

A study of the economic costs of sulfur dioxide and fluoride pollution in Montana recently has been completed (Otis et al., 1978). This study attempts to estimate the value of the damage to health, vegetation, materials and visibility. The costs were estimated for the present levels of pollution and at the levels required by the existing Montana ambient rule. Estimates also were made at the level of the federal primary ambient air quality standard for sulfur dioxide.

Health effects costs for sulfur dioxide air pollution were estimated from dose-response functions developed from statistical studies of death rates in different U. S. cities with varying levels of pollution. These were then applied to measured sulfur dioxide levels in Montana. Otis _et al_. concluded that meeting the federal annual standard of 0.03 ppm sulfur dioxide would save health costs of $6,600,000 each year. Meeting a standard of 0.02 ppm would result in annual savings of $14,400,000.

The economic effects on reduced timber growth and lower yields from wheat and other crops were estimated by an approximate, but standard, technique that assigns each county to a "pollution potential" class and defines a percentage of each crop that is lost due to airborne sulfur dioxide in counties of that class. This technique has produced results reasonably close to actual field-by-field observed losses in some instances. Otis _et al_. estimated that achieving a 0.03 ppm annual average level would save $800,000 in vegetation damage each year in Montana. The annual savings anticipated from meeting a 0.02 ppm standard were estimated to be approximately $1,000,000.

Damage to building materials, fences, paint, and other property was estimated using dose-response functions found in laboratory experiments. Otis _et al_. calculated that annual damage losses due to materials damage could be reduced by about $300,000 by meeting a 0.03 ppm annual standard and by $500,000 by meeting a 0.02 ppm annual standard.

The value to residents of Billings of the improved visibility that could be expected to result from meeting the sulfur dioxide standard was also calculated. Reduced emissions of sulfur dioxide would reduce the amount of airborne particulates obscuring visibility in the air near Billings. Otis _et al_. estimated that Billings' residents would be willing to pay $700,000 for the improved visibility that could be achieved by meeting a standard of 0.02 ppm for sulfur dioxide.

Estimates were also made of the environmental damages around the Anaconda Aluminum plant in Columbia Falls and the Stauffer Chemical phosphorous plant near Ramsay. The present control program at Anaconda Aluminum is expected to result in an approximate $1 million per year damage reduction to Glacier National Park, the Flathead National Forest and to neighboring private property. The reductions in fluoride emissions at Stauffer Chemical necessary to meet the existing Montana fluoride ambient rule are estimated to result in annual savings of about $200,000 per year just from the damage reduction to alfalfa.

During 1977 industries in Montana spent about $30 million on new air pollution control equipment. It has been estimated that approximately $15 million of this was spent in Montana for labor and an additional $5 million was spent locally for materials and equipment (DHES, 1978). These expenditures will reduce air pollution for many years. If this cost is distributed across a normal depreciation period, the annual cost of this control equipment is more nearly $2 million a year.

New pollution control equipment is subject to special tax provisions. Federal tax laws permit substantial tax credits and write-offs. These permit the recovery of 30 to 50 percent of the cost of the control equipment. In Montana special provisions of the property tax laws allow new air pollution control equipment to be taxed at about one-fourth the rate of other new plant equipment. This reduces the cost of installing pollution control equipment, but does not significantly reduce the tax income to the local community.

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V. CONCLUSIONS

This chapter briefly reiterates the documents which the Air Quality Bureau staff believes to be the most important in determining what standards should be set. The rationale for setting each standard follows the reiteration, and culminates with the standards proposed.

V.A. SULFUR DIOXIDE

Sulfur dioxide and its oxidation products have been associated with adverse health effects and adverse effects on vegetation. Sulfur oxides irritate the throat and lung and contribute to increased respiratory disease. Injury to tissues in the throat and lung (Asmundson *et al*., 1973) has been observed. The growth and yield of timber, wheat, oats and other agricultural crops important to Montana is reduced by exposure to sulfur oxides.

A One-Hour Standard

Short-term exposures to sulfur dioxide have been demonstrated to result in discomfort and impairment of lung functioning in healthy young men at levels of 0.75 to 1.0 ppm (Snell and Luchsinger, 1969; Bates and Hauzucha, 1973; Andersen *et al*., 1974).

An exposure of pine seedlings to 0.5 ppm sulfur dioxide for 30 minutes was found to inhibit growth, potentially interfering with the ability of the forest to regenerate (Constantinidou *et al*., 1976). White *et al*. (1974) found that exposure of alfalfa to 0.35 ppm sulfur dioxide for one hour also interfered with growth.

A safety factor of at least two is necessary to insure that the public health will be protected. Therefore, the Air Quality Bureau proposes a sulfur dioxide one hour ambient air quality standard of 0.4 ppm, not to be exceeded more than once a year, to protect the public health. This standard will not fully protect vegetation from injury.

A 24 Hour Standard

An increased number of deaths is observed when daily average levels of sulfur dioxide are above 0.25 ppm in the presence of airborne particulates (Lawther, 1963). Increased respiratory illness among elderly bronchitis patients also has been observed when daily average sulfur dioxide levels exceed 0.08 ppm in the presence of particulates (Lawther _et al._, 1970). Asthmatics living near a coal-fired power plant reported an increased frequency of asthma attacks when sulfur dioxide levels rose above 0.07 ppm (Cohen _et al._, 1972).

Studies of several forest plants, including ponderosa pine, have shown that significant visible injury can be expected when average daily average concentrations of sulfur dioxide exceed 0.15 ppm (Scheffer and Hedgecock, 1955; O'Gara, 1922).

Studies presented in Section IV. C clearly indicate that significant and substantial adverse effects on human health and the environment can be expected when 24 hour average concentrations of sulfur dioxide are in the range of 0.15 to 0.25 ppm. Some studies have indicated that adverse effects may be observed at levels as low as 0.07 to 0.08 ppm, especially in the presence of particulates. Other studies have indicated no threshold of effect.

Considering all the studies presented, the Air Quality Bureau proposes a 24 hour ambient air quality standard for sulfur dioxide of 0.10 ppm, not to be exceeded more than once a year to protect plants and human health. This standard may not contain any margin of safety.

An Annual Standard

An increase in the annual death rate was observed by Wicken and Buck (1964) when annual average sulfur dioxide levels were at .04 ppm in the presence of particulate. Increased respiratory illness among childred has been observed at annual average sulfur dioxide concentrations of .05 ppm in the presence of particulates (Douglas and Waller, 1963).

An annual average sulfur dioxide concentration of 0.03 ppm was found to cause a significant reduction in timber yield in pines (Linzon, 1971). A 15 percent reduction in the yield of wheat was observed in association with a 0.015 ppm annual average sulfur dioxide exposure (Guderian, 1977). Significant damage to lichen species vital to long-term survival of ecosystems has been observed at annual average exposures of 0.01 to 0.02 ppm (LeBlanc *et al.*, 1972).

Applying safety factor of two to sulfur dioxide concentrations found to be hazardous in the epidemiological public health studies, indicates the need for an annual ambient sulfur dioxide standard of 0.02 ppm. The bureau proposes this standard to protect the public health. Some growth loss to vegetation and disruption to ecosystems in Montana may be observed at this level.

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V.B. SUSPENDED PARTICULATE

Various characteristics of different types of particulate matter are associated with adverse health effects. Particles as large as 15 micrometers can be inhaled. During light exercise as much as 35 percent of the 10 micrometer particles will enter the windpipe (Task Group on Lung Dynamics, 1966).

In two studies with laboratory animals, neither sulfur dioxide nor inert particles were observed to have any effect when administered alone, but when the two were administered together with the concentration of each held the same as before, cell injury (Asmundson *et al*., 1973) and inhibition of natural protective mechanisms in the lung (Rylander, 1971) were observed. Particulate sulfates (Amdur, 1966) and sulfuric acid mist (Amdur, 1978) also have been observed to affect laboratory animals substantially more than sulfur dioxide at similar concentrations.

Several studies have observed adverse health effects associated with high daily average sulfur dioxide and particulate exposures. Lawther *et al*. (1970) recorded increased illness among elderly bronchitis patients when daily average particulate levels were above 204 ug/m^3 as measured by the British Smoke (BS) method. Van der Lende *et al*. (1975) found decreased lung function among adults when the daily particulate levels were above 88 ug/m^3 BS. Cohen *et al*. (1972) observed increased attacks among asthma patients when daily particulate levels were above 150 ug/m^3 as measured by the high-volume (TSP) method.

Adverse health effects have been observed among children where annual average particulate levels were above 132 ug/m^3 BS in the presence of sulfur dioxide (Douglas and Waller, 1964). In a study designed to observe possible health effects where sulfur dioxide exposures were very low, increased respiratory diseases (Hammer, 1977) and impaired lung functioning (Chapman, et al., 1976) were observed in a city with an annual particulate level of approximately 130 ug/m^3 TSP compared this type effects in a city with annual particulate levels of approximately 75 ug/m^3 TSP.

A 24-Hour Standard

Although there is not a direct equivalence between results obtained by the British Smoke sampling method and results obtained by the high-volume sampling method, the studies presented here indicate that decreased lung function and other adverse effects may be observed when daily ambient suspended particulate levels are above 200 ug/m^3. The Air Quality Bureau proposes a 24-hour suspended particulate ambient air quality standard of 200 ug/m^3 not to be exceeded more than once a year to protect the public health.

An Annual Standard

The studies by Hammer (1977) and Chapman et al. (1976) indicate that adverse effects to health may be observed where particulate levels exceed 130 ug/m^3 annual average. Therefore the Air Quality Bureau proposes an annual average of 75 ug/m^3 to protect the public health. This is below the level at which effects have been observed and the use of both a daily and annual standard should present an adequate margin of safety.

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V.C. SETTLED PARTICULATE

One of the most obvious nuisances from air pollution is the settling of particulate matter on material surfaces. In an extensive interview of residents of Birmingham, Alabama, Stalker and Robison (1967) found that one-third of the residents expressed an annoyance with air pollution when the settled particulate exceeded 10 gms/m^2 per month.

Based on this study, the Air Quality Bureau staff recommends a settled particulate standard of 10 gm/m^2 averaged over 30 days.

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V.D. LEAD

Lead causes several adverse health effects in humans. Children, the unborn, and pregnant women are considered the most vulnerable to adverse effects from lead exposure. The onset of various health effects of lead is known to occur at certain well defined concentrations of lead in the blood, measured in micrograms of lead per deciliter of blood (ug/dl).

Symptoms of anemia are first noted at blood lead levels of 40 ug/dl in children (Pueschel _et al_., 1972). A decrease in components of hemoglobin formation is found at levels as low as 15 to 20 ug/dl (Roels _et al_., 1976).

At 80 to 100 ug/dl, the effects of lead in the central nervous system are manifested as irreversible brain damage (Bradley and Baumgartner, 1958). At lower levels there are deficiencies in mental performance. Landrigan _et al_. (1975) found that children with blood lead levels of 40 ug/dl had lower scores on intelligence tests than children with normal blood lead. Needleman _et al_. (1978) also noted that children with 35 to 40 ug/dl blood lead performed less well on several IQ tests than those with a blood lead average of 24 ug/dl.

Lead concentrations in the ambient air can contribute to increases in blood lead through contamination of soil. Yankel _et al_. (1977) developed a method for estimating the number of children expected to exceed a blood lead level of 40 ug/dl at certain levels of lead in the air and soil. Table III.B-III on page 122 shows these relationships.

Blood lead had been sampled in children living near lead sources in Montana. The most thorough study, done in East Helena, found that seven percent of the children tested had more than 40 ug/dl blood lead (Eden, 1977).

The applicability of the Yankel study to the East Helena situation makes it extremely important in determining what ambient air lead standard should be set for Montana. Other data described above indicates that a blood lead level of 40 ug/dl cannot be safely exceeded in children.

Recorded soil lead levels of 4000 ug/g in parts of East Helena was a determining factor in formulating the standard. Applying the soil lead level of 4000 ug/g found in parts of East Helena to the formula developed by Yankel et al reveals that an air lead concentration of 2.0 ug/m^3 would result in ten percent of the children exceeding 40 ug/dl blood lead. This is reasonably close to the seven percent of the East Helena children found to have blood lead exceeding 40 ug/dl. Blood lead in East Helena children may have been artificially low because of a considerable snow depth that reduced children's contact with contaminated soil.

In view of the above data, the Air Quality Bureau staff proposes a 1.5 ug/m^3 ambient air lead standard, a calendar quarter. This restriction is expected to keep blood lead levels below 40 ug/dl in 92 percent of East Helena children living in areas where soil lead levels reach 4000 ug/g. In areas where soil lead levels are 500 to 1000 ug/g, the standard would protect 95 to 97 percent of the children from 40 ug/dl blood lead. The 90 day averaging period is based on the approximate time needed for airborne lead to cause changes in blood lead concentrations. A standard lower than 1.5 ug/m^3 would not necessarily protect every child from incurring blood lead levels of 40 ug/dl or more. The proposed standard reflects the understanding that in areas of high soil lead much of the lead found in children's blood results from contamination of soil and dust by airborne lead. The tendency of children to place dirt and other foreign matter in their mouths leads to the consumption of lead that originated as an industrial emission into the air. Once lead is deposited in soil, it remains a hazard for a long time.

A standard restricting the amount of lead in the air thus is only a partial solution to the threat of lead poisoning in children. In some areas emission of lead into the air over long periods has contaminated soils to the point that there would be a continuing hazard to children even if all emission of lead into the atmosphere were stopped. In these areas soil treatment (chelation) may be the only remedy to ensure protection of all portions of population.

The measurement method for lead is found in Appendix E.

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V.E. CARBON MONOXIDE

Carbon monoxide is absorbed into the blood through the lungs, forming carboxyhemoglobin, which interferes with the ability of the blood to transport oxygen. There is a definite correlation between any given concentration of carboxyhemoglobin in the blood and various concentrations of carbon monoxide in the air, the duration of exposure, the weight of the individual and the amount of exercise and activity during the exposure. An equation developed by Coburn, et al. (1965) demonstrates these relationships. The adverse health effects of carbon monoxide have conclusively been shown to be directly related to the concentration of carboxyhemoglobin in the blood.

The effects of carbon monoxide on general populations are evident at levels of 4 to 5 percent blood carboxyhemoglobin. At these concentrations mental processes are adversely affected (Halperin, et al., 1959; Horvath, et al., 1971; Schulte, 1963). The ability to perform physical exercise or work also is decreased at concentrations of carboxyhemoglobin in the blood above 4 percent (Aronow and Cassidy, 1975; Ekblom and Huot, 1972; Chevalier, et al., 1966).

Persons with an impaired heart, lungs or blood vessels are particularly sensitive to the effects of carbon monoxide. Carbon monoxide has been shown to cause structural changes in heart artery tissue that can lead to heart disease (Thomsen and Kjeldsen, 1974) and delay the recovery from heart attacks (DeBias, et al., 1978). Decreased tolerance for exercise has been observed at 3 percent blood carboxyhemoglobin in persons with heart disease, lung disease, or certain diseases of the blood vessels (Anderson, et al., 1973; Aronow and Isbell, 1973; Aronow, et al., 1974; Aronow, et al., 1977).

Pregnant women also are particularly sensitive to carbon monoxide. Elevated carboxyhemoglobin levels in pregnant women has been shown to correspond to lower birth weights. Very little data is available on the longer term effects on the children or the specific levels of carboxyhemoglobin causing the effect (Astrup, *et al.*, 1972).

An Eight-Hour Standard

Because of the considerable size of the populations at risk, a safety factor of 2 to 3 is justified. Thus, an ambient air quality carbon monoxide standard should prevent ambient carbon monoxide concentrations high enough to generate carboxyhemoglobin concentrations above 1.5 percent.

From Table III.C-I it can be seen that a carboxyhemoglobin level of 1.5 percent in a health woman corresponds to a 9 ppm carbon monoxide exposure for eight hours. The Air Quality Bureau proposes a 9 ppm air quality standard for carbon monoxide, averaged over 8 hours.

A One Hour Standard

Table III-C-I also shows that the body of a person engaged in heavy exercise or work will absorb carbon monoxide much more quickly than if the person is at rest. When the federal ambient air quality standard for carbon monoxide was initially adopted by the EPA it was based on a similar calculation for an adult male at rest. It is more reasonable to assume that individuals may be exposed to carbon monoxide while they are exercising or working over a one hour period. The Air Quality Bureau therefore proposes a 17 ppm air quality standard for carbon monoxide, averaged over one hour. This is approximately half the present federal standard for one-hour exposures.

The measurement method for carbon monoxide is found in Appendix E.

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V.F. NITROGEN DIOXIDE

Nitrogen dioxide is an industrial pollutant capable of causing health problems in humans at low ambient levels. It has also been shown to damage the type of plants grown in Montana.

Clinical studies have shown that the effects of nitrogen dioxide are most pronounced in children and individuals with asthma or chronic lung disease. Orehek et al. (1976) and others have found that when drugs which simulate an asthmatic attack are administered in conjunction with small amounts of nitrogen dioxide, the effects are greatly heightened, indicating nitrogen dioxide has a role in elevating such attacks under normal conditions.

Animal studies indicate that exposure to 0.5 ppm nitrogen dioxide in laboratory animals with a bacterial infection will increase the death rates (Coffin et al., 1976; Ehrlich and Henry, 1968). This results from the destruction of the antibacterial cells in the lung after corresponding to nitrogen dioxide exposure (Vosin et al., 1977).

Increased susceptibility to lung infections in humans also has been observed in several community health studies following nitrogen dioxide exposures. Increased respiratory disease has been observed in children at annual average nitrogen dioxide exposures above 0.08 ppm (Shy et al., 1970a, 1970b; Pearlman, 1971). A comparison of illness rates in homes with gas ranges and homes with electric ranges found a significantly greater incidence of cough, bronchitis, and colds going to the chest in the gas range homes (Melia et al., 1977). It was suggested that this could be due to the higher levels of nitrogen dioxide in the homes with gas stoves. Other studies found that levels of nitrogen dioxide in homes with gas stoves varied from 0.05 to 0.07 ppm, while homes with electric stoves recorded values of 0.01 ppm (Melia et al., 1978; Palmes et al., 1977).

Exposure to nitrogen dioxide at concentrations above 0.15 ppm has been observed to cause damage to various crop types grown in Montana (Sperings, 1971; Taylor and Eaton, 1966).

A One-Hour Standard

Animal studies suggest that multiple short-term exposures at levels of approximately 0.5 ppm nitrogen dioxide can increase the incidence of respiratory disease. Studies with asthma patients indicate that adverse effects may be apparent at levels as low as 0.11 ppm (Orehek *et al.*, 1976). While these studies raise serious concerns, their implications are not clear. Other studies imply effects to children from multiple exposures near 0.07 ppm nitrogen dioxide (Melia *et al.*, 1977, 1978).

The uncertainty in the information on the health effects of nitrogen dioxide requires that a margin of safety be used in arriving at an ambient air quality standard. Therefore, a nitrogen dioxide ambient air quality standard of 0.17 ppm, not to be exceeded more than once a year, is proposed. This standard is based on a safety factor of 3 applied to 0.5 ppm as a level known to be related to adverse effects. A factor of three was chosen in response to questions raised by the Orehek and Melia studies indicating that lower levels of nitrogen dioxide may cause adverse health effects under certain conditions. The standard will also protect vegetation from damage by nitrogen dioxide.

An Annual Standard

The effects observed by Shy *et al.* (1977a, 1977b) and Pearlman (1971) are the basis for the proposed annual average for nitrogen dioxide. These studies document the definite presence of adverse health effects at an annual average of 0.08 ppm nitrogen dioxide. These effects are clinically diagnosed illness, rather than a response threshold. It is clear from these studies that a standard below 0.08 ppm is essential for protection of public health. Therefore, the Air Quality Bureau proposes an annual average of 0.05 ppm for nitrogen dioxide. This level is below the level at which adverse effects have been observed, and in combination with a one-hour standard should provide an adequate margin of safety.

The measurement technique for nitrogen dioxide is found in Appendix E.

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V.G. PHOTOCHEMICAL POLLUTANTS

The term "photochemical pollutants" comprises a group of gases formed from the interaction of sunlight and certain precursor pollutants in the air. Photochemical pollutants are collectively referred to as oxidants, of which ozone is the major component. Control of oxidant pollution is accomplished through an ambient standard for ozone.

The scientific information pertaining to ozone shows that individuals with lung ailments and healthy people engaged in moderate to strenuous exercise are the most sensitive to adverse effects from ozone. It was found also that ozone alone or combined with other pollutants damages the types of crops and plant ecosystems important to Montana's welfare.

Laboratory studies found that ozone causes increased airway resistance and a decreased resistance to bacteria in the lungs of experimental animals at levels from 0.07 to 1.0 ppm (Watanabe et al., 1973; Coffin and Blommer, 1970; Miller et al., 1978).

Evidence from both clinical and epidemiological studies shows that healthy adults and people with asthma are adversely affected at short-term ozone levels between 0.15 and 0.25 ppm ozone. Shoettlin and Landau (1961) observed that asthma attacks increased at a daily average of 0.25 ppm ozone. Hammer et al. (1974) observed increased headache and eye irritation at hourly ozone averages of 0.15 ppm.

Decreases in lung function have been recorded in healthy adults exposures of 0.25 to 0.37 ppm ozone over two hours (Hazucha et al., 1973; Hackney et al., 1975). At 0.15 ppm ozone individuals engaged in moderate exercise show similar responses (Delucia and Adams, 1977). It also is suggested that maximum physical activity decreases the amount of ozone necessary to affect performance to levels as low as 0.05 ppm (Wayne et al., 1969).

Plants found in Montana are affected by 0.05 to 0.10 ppm ozone over various exposure periods (Neeley et al., 1977; Wilhour and Neeley, 1977). Ozone in combination with other pollutants causes plant damage at lower levels than when it is alone (Kress and Skelly, 1977; Bennett and Hill, 1974). Ecosystem structure and function also is known to be reduced from ozone exposure (EPA, 1977).

A One Hour Ozone Standard

Studies clearly show that 0.15 to 0.25 ppm ozone decreases lung function in healthy subjects, at rest or moderately exercising. At 0.15 ppm ozone, other ailments such as headaches and eye irritation increase. Considering these facts, the Air Quality Bureau proposes a 0.10 ppm one hour ozone standard, not to be exceeded more than once a year, which would keep concentrations low enough to protect health. The proposed standard is based on a 0.15 ppm threshold level for adverse impact with a 1.5 margin of safety. The margin of safety necessary for Montana was determined to be lower than that necessary for protection of human health in warmer and sunnier areas where conditions for ozone production are generally more favorable. The proposed standard also would protect Montana's vegetation from ozone/oxidant damage except when ozone was combined with large amounts of other pollutants.

The measurement technique for ozone is found in Appendix E.

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V.H. VISIBILITY

The "big sky" is an important part of the state heritage for most Montanans. An ambient air quality standard for visibility would help protect that heritage.

Visibility observations at airports during recent years indicate that the annual average visibility in eastern Montana is approximately 60 to 70 miles, depending on the specific location. In the western part of the state, the average is lower, due to higher humidity, more precipitation, and other adverse weather (Trijonis and Shapland, 1978).

At a minimum, an ambient air quality standard for visibility should prevent any further degradation in the visibility Montanans enjoy. To establish a uniform, state-wide air quality standard, it is necessary to use a measurement method that will compensate for the higher humidity that contributes to lower average visibility in the western part of the state. Therefore, the Air Quality Bureau proposes a visibility standard of 2×10^{-5} per meter particle scattering coefficient as measured by the integrating nephelometer method described in Appendix E. This standard establishes an annual average visibility that will ensure no visibility degradation over that now experienced.

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V.I. FLUORIDE

Human health does not appear to be the determining factor in establishing an ambient air quality standard for fluoride. Rather, the critical determinant is the effects of fluoride on plants and animals. Among the common air pollutants hydrogen fluoride is the most toxic to plants.

Injury to human health by airborne fluoride has not been documented at concentrations observed in Montana. However, European researchers have observed reduced hemoglobin levels and other indicators of adverse health effects in people residing in fluoride contaminated areas (Balazova *et al.*, 1970; Leloczky, 1971; Rippel, 1967). Fluoride levels in the air were not reported in these studies making it impossible to accurately compare the European studies with conditions in Montana. These studies do suggest that monitoring the health of persons living near fluoride-emitting sources in Montana appears desirable.

The degree of fluoride damage in plants is determined by the total accumulation of the substance in plant tissue. That is, a critical level for irreparable damage can be caused by short-term high concentrations or longer exposures at lower concentrations. The most common indicator of plant damage is a type of leaf injury characteristically resulting from excessive fluoride. This type of visible injury causes reductions in growth and yield of plants and ultimately death if fluoride exposure is severe enough. It is important to note that growth and yield losses have also been found even when there is no visible fluoride injury. A number of timber species found in Montana are particularly sensitive to fluoride pollution.

Both gaseous and particulate fluoride pollution accumulates in or on vegetation. Fluoride in vegetation becomes available to grazing animals for consumption. A number of studies have shown that certain levels in plant material consumed by livestock can cause abnormalities in developing teeth and bones. Higher levels can reduce weight and milk production. Other animals, such as small mammals and insects, also have been shown to acquire excessive fluorides from consumption of contaminated plant materials.

A Fluoride Forage Standard

The potential effects from high fluoride in livestock forage is well accepted in the scientific community (NAS, 1971). Suttie (1969) wrote that a forage fluoride standard is the only practical means of protecting livestock from excessive fluoride in forage.

Various controlled feeding experiments conducted under laboratory conditions indicate that forage fluoride levels in excess of 40 ppm (ug/g) causes economic damage to livestock with continued feeding (Suttie *et al.*, 1957; Greenwood *et al.*, 1964).

Field experiments conducted in Montana and elsewhere suggest that low levels of fluoride in plant tissue can cause adverse effects in cattle and livestock (Miles *et al.*, 1978; Krook and Maylin, 1978). The work of Krook and Maylin (1978) shows that animals pastured near fluoride sources are exposed to fluoride continually from before birth until slaughter. Their work found severe tooth injury, lameness, and growth loss at seasonal fluoride in forage averages were below 32 ug/g. It also suggested that bone fluoride levels are not the best indication of fluoride damage to livestock. Perhaps more important is the time of exposure, with young animals and fetuses being the most sensitive.

The evidence available indicates that livestock and wildlife are adversely affected after long-term consumption of forage with less than 40 ug/g fluoride.

Laboratory feeding experiments usually predict injury levels lower than those actually occurring because they do not reflect natural feeding habits, or nutritional and environmental stresses imposed on pasture and range animals. Also, they do not consider fluoride effects on fetuses and newly born calves, when bone development is most sensitive to fluoride exposure. Fluoride dosage in field experiments is never controlled as rigorously as in laboratory experiments, but it is clear that livestock injury occurs at lower forage fluoride levels under field conditions than in controlled laboratory studies. The most comprehensive field study (Krook and Maylin, 1978) indicates damage to livestock occurs at a level no higher than 32 ug/g fluoride. The Air Quality Bureau staff recommends a standard limiting fluoride in forage to 30 ug/g (dry weight basis) not to be exceeded.

A 24-Hour Hydrogen Fluoride Standard

The need for a short-term hydrogen fluoride standard is suggested by several experiments with Montana plants that indicate either stress or direct injury after 24 to 48 hours of exposure. The experiments of Applegate and Adams (1960) with bush beans indicate that 14 ppb hydrogen fluoride for a 24-hour exposure causes increased plant respiration, indicating the plant was under stress. Solberg *et al*. (1955) found that significant numbers of ponderosa pine exposed to 1.5 ppb for 48 hours showed visible signs of injury. Similarly, Adams *et al*. (1956a) observed that ponderosa pine exposed to 1.22 ppb hydrogen fluoride for 24 hours showed slight injury to their needles.

Continuing stress and visible injury in plants eventually will result in decreases in growth and yield of the affected plants. Therefore, exposure levels below those observed in the above experiments are required to protect vegetation. The Air Quality Bureau staff recommends setting a standard of

1.0 ppb 24-hour average not to be exceeded more than once per year. The observation of Treshow et al. (1967) that growth reductions are possible even in the absence of visible signs of injury suggests that this standard may not prevent all economic losses from fluoride exposure but there is no experimental work available to support a more stringent standard.

A 30-Day Standard for Hydrogen Fluoride

Two lines of persuasion suggest a 30-day fluoride standard. The first relates to experiments showing direct adverse effects to plants at or near a 30-day averaging time. The second relates to the degree of hydrogen fluoride exposure necessary to cause a 30 ppm accumulation of fluoride in forage, posing a hazard to plant eating animals.

Solberg et al. (1955) found that a 0.50 ppb hydrogen fluoride exposure for nine days caused visible injury to the needles of ponderosa pines tested under laboratory conditions. Adams et al. (1956b) found that 0.49 ppb for 15 days caused similar injury to ponderosa pine exposed under field conditions. Finally, Sidhu (1977) found that four trees species grown in Newfoundland were damaged by hydrogen fluoride at: 0.55 ppb/31 day for balsam fir; 0.48 ppb/26 days for eastern larch; and 0.50 ppb/20 days for white birch and alder. These trees are considered more resistent to hydrogen fluoride than western forest trees (Weinstein, 1977).

Injury to livestock and wildlife occurs when fluoride exceeds 30 ug/g in forage. The average atmospheric fluoride concentration that causes an increase of 30 ug/g fluoride in plant tissue has been calculated for a number of Montana forage crops as shown on page 197. The work of Guderian (1969) illustrates the range of concentrations that could yield 30 ug/g fluoride in eight forage crops. Average ambient air concentrations ranged from 0.94 ppb to 0.31 ppb. All values were calculated from laboratory exposure studies. The work of Israel (1974) conducted under field conditions indicates an average 30-day hydrogen fluoride level of 0.22 ppb is capable of causing 30 ug/g fluoride accumulation in two important forage crops grown in Montana.

Direct exposure studies provide evidence that sensitive western forest species are damaged by hydrogen fluoride at approximately 0.50 ppb for 9 to 15 days exposure. Moderately tolerant eastern forest species are damaged at approximately 0.50 ppb hydrogen fluoride for 20 to 31 days of exposure. This data suggests that Montana forest species require a standard below 0.50 ppb, 30 day average. Accumulation experiments suggest that Montana forage crops will accumulate 30 ug/g fluoride at 30-day average hydrogen fluoride exposures between 0.94 ppb and 0.22 ppb.

Considering this information, the Air Quality Bureau staff recommends an air quality standard at 0.30 ppb hydrogen fluoride averaged over 30 days not to be exceeded more than once a year to protect sensitive forest trees from visible fluoride injury and to protect forage crops from excessive fluoride accumulation. This standard does not reflect consideration of particulate fluoride accumulation in plants.

A Growing Season Standard for Hydrogen Fluoride

Fluoride accumulates in plant ecosystems through one or more growing seasons. Work by Sidhu (1977 and 1978) in Newfoundland and Carlson (1978), Kay (1974), and Miles *et al*. (1978) in Montana has shown this trend in field studies near fluoride emitting facilities. The study conducted by Carlson and Dewey (1971) is particularly significant since it relates fluoride accumulation to injury in several Montana plant species.

Carlson and Dewey (1971) found that 15 percent of plots containing a number of trees, shrubs, and herbs near the Anaconda Aluminum facility at Columbia Falls had visible injury and an average fluoride content of 16 ug/g fluoride. An EPA (1974) air monitoring program showed an average hydrogen fluoride level during this time period to be between 0.13 and 0.16 ppb. Similarly, at an average hydrogen fluoride level between 0.16 and 0.12 ppb, Carlson and Dewey found 46 percent of the plots with visible injury while accumulating 34 ug/g plant fluoride.

The Air Quality Bureau recommends an air quality standard of 0.13 ppb hydrogen fluoride as a growing season standard. This proposal would limit growing season hydrogen fluoride average concentrations to the lowest value in the range found to damage forests in Montana. It is also intended to reduce long-term fluoride accumulation in plant ecosystems.

Economic Analysis

An economic analysis (Otis *et al.*, 1978) of fluoride damage to timber and other crops near the two most important sources of fluoride air pollution in Montana estimated the value of lost agricultural production and the cost of the control equipment necessary to meet these standards. The projected savings to society were approximately the same or greater than the net costs to the industry of meeting these standards. Even without considering the tendency of these economic studies to substantially underestimate the social costs of pollution, the standards appear to be of net economic benefit to the state.

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V.J. HYDROGEN SULFIDE

Community objections to the odor of hydrogen sulfide has resulted in efforts on the part of most industrial sources to control their emissions of hydrogen sulfide. No community health surveys have been conducted to determine what level are low enough to have no effect on public health. Community exposures to hydrogen sulfide sufficient to cause observable health effects occur only as a result of an industrial accident or other abnormal circumstance. In one instance where ambient concentrations were documented during an accidental community exposure, reports of adverse reactions were received from areas with concentrations of 0.3 ppm (Public Health Service, 1964).

Adverse effects have been observed on several crops and timber species at hydrogen sulfide concentrations of 0.3 ppm and on Douglas fir at 0.1 ppm (Thompson and Kats, 1978).

The average threshold for detection of the odor is 0.0045 ppm (Wilby, 1969). However there have been no studies to establish a concentration sufficient to cause an odor strong enough to be a public nuisance. The strong reaction of the public to hydrogen sulfide indicates the need for a safety factor of 3. The Air Quality Bureau proposes a one hour standard of 0.1 ppm, to protect the public health.

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APPENDIX A.

MONTANA AIR QUALITY DATA

TABLE A-I. BILLINGS AREA ESTIMATED EMISSIONS - 1977

Sources	Estimated Emissions - (Tons Per Year) Sulfur Dioxide	Particulates
Point Sources		
Cenex - Laurel	10,380	398
Conoco - Billings	3,198	263
Exxon - Billings	9,800	932
Great Western Sugar Co. - Billings	815	65
Montana Power Company - Billings	9,986	1,124
Montana Sulphur & Chemical Co. - Billings	1,530	Neg.
U. S. Gypsum - Lewistown	Neg.	73
Westmoreland - Hardin	---	19
Area Sources*		
Unpaved Roads	---	141,786
Agriculture	---	5,839
Open Burning	---	1,125
Other	2,007	3,662

*Only the counties of Yellowstone, Carbon, Stillwater, Sweetgrass and Big Horn

Source: Gelhaus *et al*. (1978)

TABLE A-II. EASTERN MONTANA ESTIMATED EMISSIONS - 1977

Sources	Estimated Emissions - (Tons Per Year) Sulfur Dioxide	Particulates
Holly Sugar Company - Sidney	226	116
Montana Power Co. Unit #1 & #2 - Colstrip	5326	618
Montana-Dakota Utilities - Sidney	2372	430
Tumpane Co. - Glasgow AFB	174	29
Area Sources*		
Unpaved Roads	---	81,170
Agriculture	---	4,068
Open Burning	---	803
Other	596	1,427

*Only the counties of Rosebud, Treasure, Custer, Fallon, Powder River and Carter

Source: Gelhaus *et al*. (1978)

TABLE A-III. GREAT FALLS AREA ESTIMATED EMISSIONS - 1977

Sources	Estimated Emissions - (Tons Per Year) Sulfur Dioxide	Particulates
Point Sources		
Con-Agra - Great Falls	0	70
General Mills - Great Falls	0	32
Phillips Petroleum - Great Falls	1,807	72
Westco Refining - Cut Bank	883	11
Area Sources	2,600	251,000

Source: Gelhaus *et al*. (1978)

TABLE A-IV. HELENA-BUTTE AREA ESTIMATED EMISSIONS - 1977

Sources	Estimated Emissions - (Tons Per Year) Sulfur Dioxide	Particulates
ASARCO - East Helena	14,500	418
Anaconda Company - Anaconda	281,750	4,780
Kaiser Cement - Montana City	---*	204
Ideal Cement	---	263
Pfizer - Dillon	5	124
Berkeley Open Pit Mine - Butte	207	4023
Stauffer Chemical Co. - Silver Bow	208	73
U. S. Plywood - Silver City	1	13
Townsend Lumber - Townsend	1	20
Yellowstone Pine - Belgrade	1	15
Elk Studs - West Yellowstone	1	12
Burkland Studs - Livingston	1	12
Area Sources+		
Unpaved Roads	---	81,278
Agriculture	---	101
Open Burning	---	7,047
Other	977	2,816

*Unknown
+Only the counties of Lewis & Clark, Deer Lodge, and Silver Bow
Source: Gelhaus et al. (1978)

TABLE A-V. MISSOULA AREA ESTIMATED EMISSIONS - 1977

Sources	Estimated Emissions (Tons Per Year) Sulfur Dioxide	Particulates
Point Sources		
Anaconda Aluminum Columbia Falls	2,200	1,440
Louisiana-Pacific (particleboard) Missoula	Neg	92
Evans Products (plywood) Missoula	24	37
W. R. Grace Libby	700	4.5
Champion-Hoerner Waldorf Missoula	365	760
Plum Creek Lumber Columbia Falls	65	34
St. Regis Paper Libby	277	696
Champion-U.S. Plywood Bonner	224	288
Area Sources*		
Unpaved Roads	---	158,386
Agriculture	---	245
Open Burning	---	13,623
Other	3,282	3,221

* For the counties of Flathead, Lake and Missoula

Source: Gelhaus et al. (1978)

TABLE A-VI. POPULATION TRENDS IN COUNTIES WITH MAJOR AIR POLLUTION SOURCES

County	Population 1970	Population, 1980*
Flathead	39460	48900
Missoula	58263	69500
Deer Lodge	15652	13500
Silver Bow	41981	40900
Yellowstone	87367	109700
Rosebud	6032	10100
Richland	9837	10400

*Projection from Department of Community Affairs, April, 1978

TABLE A-VII.
SOURCE OF CARBON MONOXIDE, HYDROCARBON AND NITROGEN OXIDE EMISSIONS FOR MONTANA

		CO	HC	NO_2
Cascade	Point Source	10,578	542	113
	Area Source	91,000	14,900	8,000
	Total	101,578	15,442	8,113
Custer	Point Source	0	0	0
	Area Source	13,300	2,100	1,200
	Total	13,300	2,100	1,200
Deer Lodge	Point Source	5,007	884	67,747
	Area Source	17,700	2,600	1,200
	Total	22,707	3,484	69,047
Lewis & Clark	Point Source	46,375	248	0
	Area Source	50,400	7,100	3,900
	Total	96,775	7,348	3,900
Missoula	Point Source	17,908	4.3	1,896
	Area Source	89,000	12,200	6,700
	Total	106,908	12,613	8,596
Silver Bow	Point Source	22	2	40
	Area Source	46,500	7,800	7,500
	Total	46,522	7,802	7,540
Yellowstone	Point Source	59,148	43,000	9,410
	Area Source	70,157	16,000	8,800
	Total	129,305	59,000	18,210

Source: Unpublished AQB data, Battelle, 1970, PEDCo, 1975.

TABLE A-VIII. AMBIENT CONCENTRATIONS OF CARBON MONOXIDE AT VARIOUS LOCATIONS

COMMUNITY AND COLLECTION SITE**	MONTH/YEAR OF COLLECTIONS		MAX. CONCENTRATIONS 1 hr.	8 hr.	VIOLATION 8 HR. STANDARD
Missoula, (High. 93, Junct.)*	March	1977	17.5	11.1	6
Butte (Alpine West)	March	1977	3.7	2.18	0
Great Falls (908 10th Ave. S.)	December	1977	23.1	14.35	1
Colstrip (McRae Ranch)	May	1975	No readings within detection limits		
Billings (11th and S. 27th St.)*	March	1976	3.9	2.29	0
Billings (Grand Ave.)*	June	1975	9.9	5.47	0

*Violations of the Federal 9 ppm 8 hr. ave. standard are recorded at least once at each of these stations over the monitoring history (Including months other than the data shown).

**Maps of monitoring sites are found on page

Source: Gelhaus et al. (1976, 1977, 1978)

TABLE A-IX. AMBIENT CONCENTRATIONS OF NITROGEN DIOXIDE IN VARIOUS MONTANA LOCATIONS

DATES	LOCATIONS					
	Billings*		Missoula*		Butte*	
	1 hr.	Annual Avg. (Year to date)	1 hr.	Annual Avg. (Year to date)	1 hr.	Annual Avg. (Year to date)
1/78	0.10 ppm	0.039 ppm				
11/77			0.09 ppm	0.02 ppm		
8/77	0.18 ppm	0.029 ppm			0.06 ppm	0.012 ppm
7/77	0.14 ppm	0.018 ppm			0.06 ppm	0.015 ppm
3/77	---	---			0.08 ppm	0.022 ppm
2/77	0.04 ppm	0.023 ppm				
8/76	0.08 ppm	0.032 ppm				
7/76	0.11 ppm	0.033 ppm				
3/76	0.10 ppm	0.041 ppm				
2/76	0.08 ppm	0.043 ppm				
1/76	0.10 ppm	0.048 ppm				

*Billings monitor located at 27th and Montana; Missoula at Lion's Park; Butte at Alpine West.

Source: Gelhaus et al. (1977, 1978)

TABLE A-X. AMBIENT CONCENTRATIONS OF OZONE AT VARIOUS MONTANA LOCATIONS

Month & Year		Total Readings	1 Hr. Periods Over: 0.02	0.04	0.06	0.08	0.10
BILLINGS							
April	1978	499	325	20	0	0	0
March	1978	725	350	65	9	4	2
February	1978	659	361	45	4	1	0
January	1978	672	273	2	0	0	0
September	1977	586	199	20	0	0	0
August	1977	579	316	61	1	0	0
July	1977	681	487	93	0	0	0
June	1977	644	396	130	4	0	0
April	1977	552	343	81	0	0	0
September	1976	645	157	10	0	0	0
August	1976	697	322	68	10	2	0
July	1976	718	522	237	32	9	1
June	1976	597	359	51	2	1	0
May	1976	660	388	23	0	0	0
April	1976	615	432	128	27	1	0
March	1976	714	470	182	0	0	0
MISSOULA							
March	1978	646	346	89	1	0	0
February	1978	659	191	40	0	0	0
January	1978	621	61	0	0	0	0
December	1978	37	6	0	0	0	0

Source: Gelhaus *et al*. (1977, 1978), Unpublished AQB data

TABLE A-XI. AGRICULTURAL PRODUCTION IN COUNTIES WITH MAJOR AIR POLLUTION SOURCES

Acres planted/yield per acre

County	Wheat* (bushels)	Barley* (bushels)	Corn** (bushels)	Sugarbeets** (tons)	Hay** (tons)	Beans** (100 lb bag)	Oats** (bushels)
Deer Lodge	500/39.8	1,800/56.9	0	0	11,200/2.07	0	0
Flathead	11,900/45.8	37,000/48	200/16+	0	35,200/2.58	0	1,000/37.3
Missoula	2,000/33.8	5,600/38.8	300/17	0	13,400/2.11	0	2,200/60.5
Lewis and Clark	10,800/23.3	14,100/47.7	100/16	0	46,700/2.06	0	4,200/70.5
Yellowstone	103,500/29.8	27,100/45.0	1,100/20	10,050/21	21,700/2.41	800/17	3,600/57.8
Rosebud	39,000/27.2	21,100/36.8	8,600/16	1,260/19	38,000/2.07	400/15	8,500/49.5
Richland	156,600/22.7	37,400/29.5	8,200/10 dry 8,200/21 irrig.	11,330/17	48,000/1.21	3,900/12	29,400/55.1

* 1977 estimate
** 1975 data

Source: Montana Department of Agriculture and Statistical Reporting Service, USDA

TABLE A-XII. AMBIENT CONCENTRATIONS OF HYDROGEN SULFIDE IN THE MISSOULA AREA

Date	No. ½ Hour Periods Over 30 ppb	No. 8 Hour Periods Over 10 ppb	Monthly Maximum
July 1977	15	2	56.1
August 1977	16	1	69.0
September 1977	0	0	23.6
October 1977	5	1	53.6 (2)
November 1977	0	0	22.8
December 1977	2	4	33.7
January 1978	0	0	23.1
February 1978	3	4	38.7
March 1978	0	2	25.0
April 1978	No Data		
May 1978	2	0	36.7
June 1978	No Data		

Source: AQB files

TABLE A-XIII. NEW POLLUTION CONTROL EQUIPMENT AT HOERNER WALDORF

Control	Pollutant	Approximate Efficiency (Percent)
Holding pond aerators	Hydrogen sulfide	90
Air Stripper	Hydrogen sulfide from condensate streams	90
Venturi scrubber on lime kiln	Particulates	99+
Scrubber on #3 smelt tank	Particulates	95
Entrainment separators on brown stock washer hoods	Particulates	80
Recovery boiler electrostatic precipitator	Particulates	99+
Indirect contact furnace	Total reduced sulfur	99+
Miscellaneous raw material handling-		
baghouses	Particulate	99+
cyclone collectors	Particulate	98

Source: AQB files

TABLE XIV.

AMBIENT CONCENTRATIONS OF FLUORIDE IN GLACIER NATIONAL PARK

(ug/cm^2/30 days)

Highest Monthly Value Recorded At Each Sampling Location

Year	1	2	3	4	5	6	7	8	9	10
1978	0.64	0.55	0.64	0.19	0.28	0.08	0.47	0.39	0.72	0.05
1977	0.64	0.58	0.44	0.16	0.08	0.14	0.67	0.34	0.56	0.47
1976	0.58	0.58	0.16	0.08	0.08	0.15	0.55	0.33	0.67	0.56

Source: Gelhaus *et al.* (1977, 1978)

AMBIENT CONCENTRATIONS OF FLUORIDE IN COLUMBIA FALLS AND VICINITY

Highest 24-hr. Value Recorded at Each Sampling Station (ppb)

Highest Monthly Value Recorded at Each Station (ug/cm^2 /28 days)

Year	0.6 NW	2.0 S	2.0 N	4.5 E	4.5 NE	0.6 NW	2.0 NW	2.0 S	2.0 N	2.0 WSW	2.5 E	4.5 SSW	4.0 SSW	4.5 NE	10.0 SSW
1976	6.46	2.37	7.08	2.78	1.26	2.20	0.85	2.31	1.14	1.42	1.23	0.91	1.12	0.47	0.44
1977	7.84	2.42	6.47	3.23	1.87	2.03	0.67	3.40	1.37	1.35	1.67	0.38	2.08	0.47	0.50
1978	4.49	2.38	5.23	2.33	1.66	2.43	1.93	2.46	0.98	1.34	2.24	-	0.94	0.52	0.90

Source: AQB files

TABLE A- XV. ANACONDA ALUMINUM COMPANY VEGETATION DATA - 1977 (ug/g Fluoride)

ate	Sample	Total Number Samples	No. Samples Above 35 ug/g	Percent Above ug/g
/77	Grass	28	0	0
/77	Grass	25	1 (48 ug/g)	4
	1977 Needles	25	0	0
0/77	1977 Needles	28	1 (49 ug/g)	4
	1976 Needles	28	4 (58-144 ug/g)	14

ource: AQB files

TABLE XVI. RANGE OF CONCENTRATIONS (in ug/g) OF FOUR HEAVY METALS IN THE SURFACE SOILS NEAR ANACONDA, MONTANA

	Total		Biochemically Available*	
	High	Low	High	Low
Arsenic	2,362	56	258	1
Copper	8,450	50	3,260	1
Lead	1,500	23	284	0
Zinc	3,100	80	880	0

* water soluble arsenic and ammonium acetate extractable copper, lead & zinc.

Source: Taskey (1972)

TABLE A-XVII. AMBIENT CONCENTRATIONS OF SULFUR DIOXIDE IN VARIOUS MONTANA LOCATIONS

Community Site	Month/Year of sample	Maximum 1-hour	Corresponding 24-hour	Annual Arith. Average (Running Year)	Comments
Anaconda (Highway Jct.)	Dec. 1976	1.58	0.49	0.03	Fed. Prim. 24-hour Std. exceeded 9 times
	Nov. 1977	>2.00	0.72	0.037	Fed. Prim. 24-hour Std. exceeded 29 times
	Mar. 1978	>2.00	0.36	0.054	Fed. Prim. 24-hour Std. exceeded 30 times
East Helena (Microwave)	June 1976	1.08	0.06	0.014	Fed Prim. 24-hour Std. exceeded once
	June 1977	1.40	0.10	0.012	Fed. Prim. 24-hour Std. exceeded once
	Feb. 1978	0.32	0.16	0.011	
Colstrip (BN)	Mar. 1976	0.02	0.00	0.00	
	Apr. 1977	0.10	0.05	0.000*	
	1978	-	-	-	
Billings (Laurel Farm)	Sept. 1976	0.80	0.20	0.014*	Fed. Prim. 24-hour Std. exceeded 38 times
	June 1977	0.63	0.06	0.071	Fed. Prim. 24-hour Std. exceeded 32 times)
	Jan. 1978	0.66	0.29	0.049	Fed. Prim. 24-hour Std. exceeded 8 times

* Less than full 12 months data

Source: Unpublished AQB data; Gelhaus *et al.* (1977, 1978)

TABLE A-XVIII. AMBIENT CONCENTRATIONS OF COMPONENTS OF SUSPENDED PARTICULATE IN VARIOUS MONTANA LOCATIONS

ug/m^3

(Sites	Period	No. Days	Total Suspended Particulate Max. (24-hr)		Sulfate Max. (24-hr)		Nitrate Max. (24-hr)		Lead Max. (24-hr)		Cadmium Max. (24-hr)		Iron Max. (24-hr)		Arsenic Max. (24-hr)	
			Max. (24-hr)	Avg.	Max. (24-hr)	Avg.	Max. (24-hr)	Avg.	Max. (24-hr)	Avg.	Max. (24-hr)	Avg.	Max. (24-hr)	Avg.	Max. (24-hr)	Avg.
Missoula (Lion's Park)	3/78- 4/78	60	676	151	13.5	3.6	12.8	2.5	1.9	.6	.007	.002	5.6	2.2	.03	.003
Anaconda (High Way Jt)	3/78- 4/78	10	94	29	10.9	6.6	2.2	.7	2.7	.5	.12	.02	6.1	1.4	1.8	.32
Butte (Greeley School)	2/78- 4/78	14	140	94	7.7	4.9	5.1	2.7	.8	.5	.01	.006	3.0	1.6	.26	.05
Billings (City Hall)	2/78-	15	175	87					.6	.4	.001	.001	5.8	2.1	.003	.001
Colstrip (Various)	5/75-	50							.04	.01			2.0	.4	.009	.003

Source: Unpublished AQB data; Gelhaus et al. (1976)

TABLE A-XIX. AMBIENT CONCENTRATIONS OF LEAD AT VARIOUS MONTANA LOCATIONS

No. of Readings	Locale	Site	Month-Year	Monthly Average ug/m^3
5	East Helena	Hastie	January 1977	2.96
4	"	"	February 1977	5.61*
5	"	"	March 1977	3.32
5	"	"	April 1977	6.10
2	"	"	May 1977	2.25
6	"	"	June 1977	2.22
5	"	"	July 1977	2.78
3	"	"	August 1977	1.26
1	"	"	September 1977	1.93
5	"	"	October 1977	4.63
4	"	"	November 1977	15.36*
2	"	"	December 1977	14.04*
3	"	"	January 1978	12.73*
9	"	"	February 1978	3.47
1	"	"	March 1978	0.64
4	"	Microwave	January 1977	0.51
2	"	"	February 1977	2.02
6	"	"	March 1977	0.04
5	"	"	April 1977	0.03
5	"	"	May 1977	0.04
5	"	"	June 1977	0.92
4	"	"	July 1977	0.03
5	"	"	August 1977	0.38
4	"	"	September 1977	0.14
4	"	"	October 1977	0.32
6	"	"	November 1977	0.35
5	"	"	December 1977	2.53
12	"	"	January 1978	2.00
10	"	"	February 1978	0.49
1	"	"	March 1978	0.31
4	Anaconda	Highway Jct.	January 1977	0.43
4	"	"	February 1977	0.34
4	"	"	March 1977	0.60
5	"	"	April 1977	0.25
6	"	"	May 1977	0.20
4	"	"	June 1977	0.41
6	"	"	July 1977	0.05
5	"	"	August 1977	0.39
5	"	"	September 1977	0.47
5	"	"	October 1977	0.49
5	"	"	November 1977	1.71
5	"	"	December 1977	0.65
5	"	"	January 1978	0.82
4	"	"	February 1978	0.24
5	"	"	March 1978	0.74
3	"	"	April 1978	0.06

*Violations of state standard
Source: Unpublished AQB data

TABLE XX. AMBIENT CONCENTRATIONS OF FLUORIDE IN RAMSAY

Quarter	Sampler Location (Miles) and Number of Readings above 1 ppb 24-hour average		
	0.6 ESE	2.0 NW	1.0 NE
2nd 1978	44	1	5
1st 1978	15	2	33
4th 1977	20	4	9
3rd 1977	17	0	1
2nd 1977	15	0	1
1st 1977	14	2	7
4th 1976	1	0	1

Source: AQB files

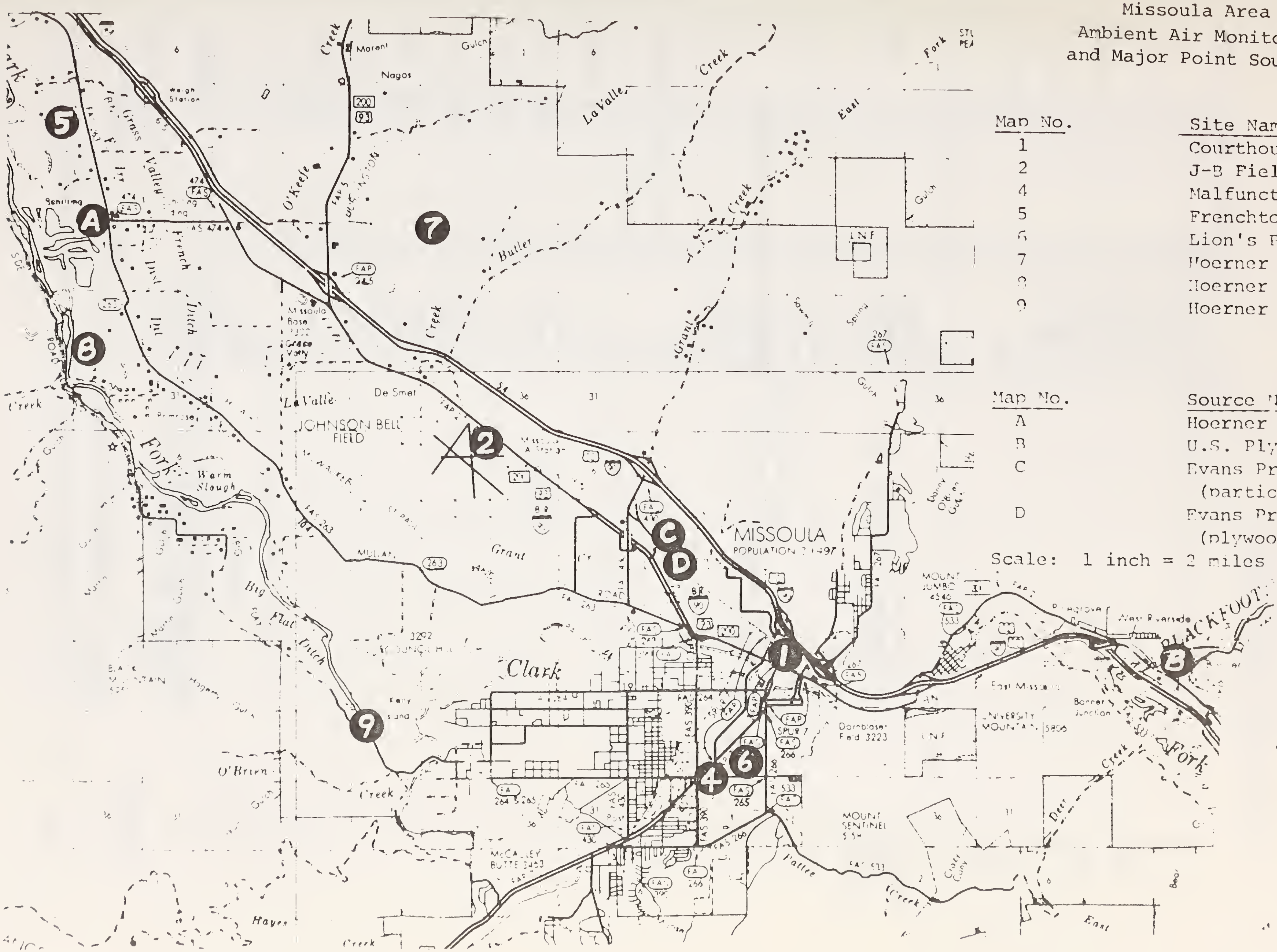
STAUFFER CHEMICAL COMPANY VEGETATION (GRASS) DATA 1976-1977
(ug/g Fluoride)

Date	Total Number Samples	No. Samples Above 35 ug/g	Percent Above 35 ug/g
11/1/76	68	24	35
7/1/77	68	1	1
9/1/77	68	10	15
11/2/77	68	22	32

Source: AQB files

FIGURE 1

Missoula Area
Ambient Air Monitors
and Major Point Sources


MISSOULA
POPULATION 2,1497
JOHNSON BELL FIELD
Clark
La Valle
De Smet
Frenchtown
Grass Valley
O'Keefe
Butler
Grant
Creek
Fork
Warm Slough
Big Flat Ditch
O'Brien
BLACKFOOT
MOUNT JUMBO 4540
MOUNT SENTINEL
UNIVERSITY MOUNTAIN 5806
East Missoula
Bonner Junction
West Riverside
McCAULEY BUTTE 3453
Nagos
Moront
1
2
4
5
6
7
8
9
A
B
C
D

Map No.	Site Name
1	Courthouse (2)
2	J-B Field
4	Malfunction Jct.
5	Frenchtown
6	Lion's Park
7	Hoerner Waldorf #1
8	Hoerner Waldorf #2
9	Hoerner Waldorf #3

Map No.	Source Name
A	Hoerner Waldorf
B	U.S. Plywood
C	Evans Products (particleboard)
D	Evans Products (plywood)

Scale: 1 inch = 2 miles

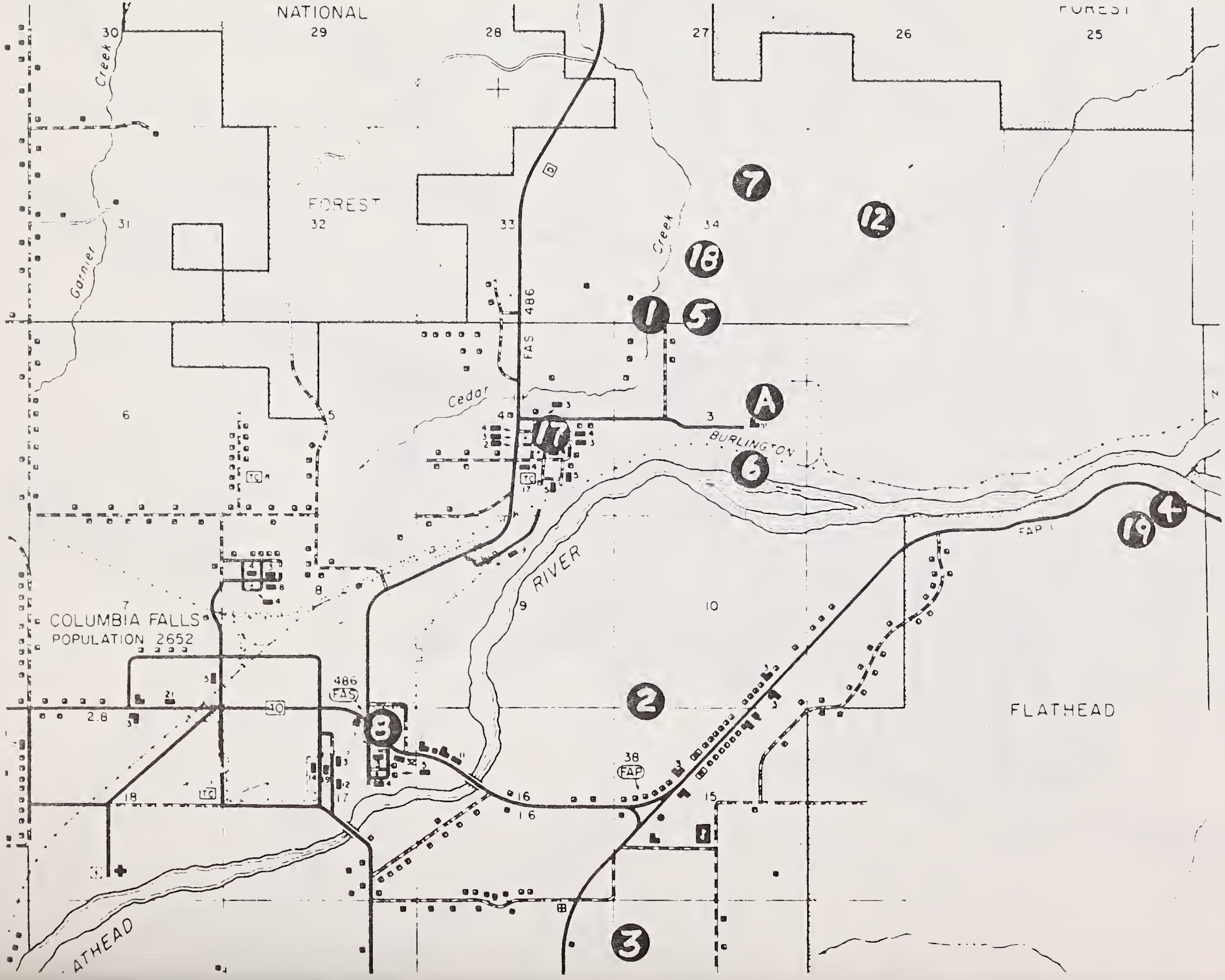

NATIONAL
FOREST
FOREST
FLATHEAD
ATHEAD
RIVER
Garnier
Creek
Cedar
Creek
BURLINGTON
COLUMBIA FALLS
POPULATION 2652
FAS 486
486 FAS
38 FAP
FAP 1
30
29
28
27
26
25
31
32
33
34
6
5
7
8
9
10
2.8
18
17
16
15
3
1
2
3
4
5
6
7
8
12
17
18
19
A

FIGURE 2
Columbia Falls Area
Ambient Air Monitors
and Major Point Source

Map No.	Site Name
1	Dehlbom Res.
2	Feirstein
3	R.E. Owen
4	Badrock
5	Dehlbom Field
6	Anaconda S
7	Teakettle #1
8	Anders Residence
17	Aluminum City
18	Trailer
19	Kenas Residence

Map No.	Source Name
A	Anaconda Aluminum

Scale: 1 inch = 0.80 miles

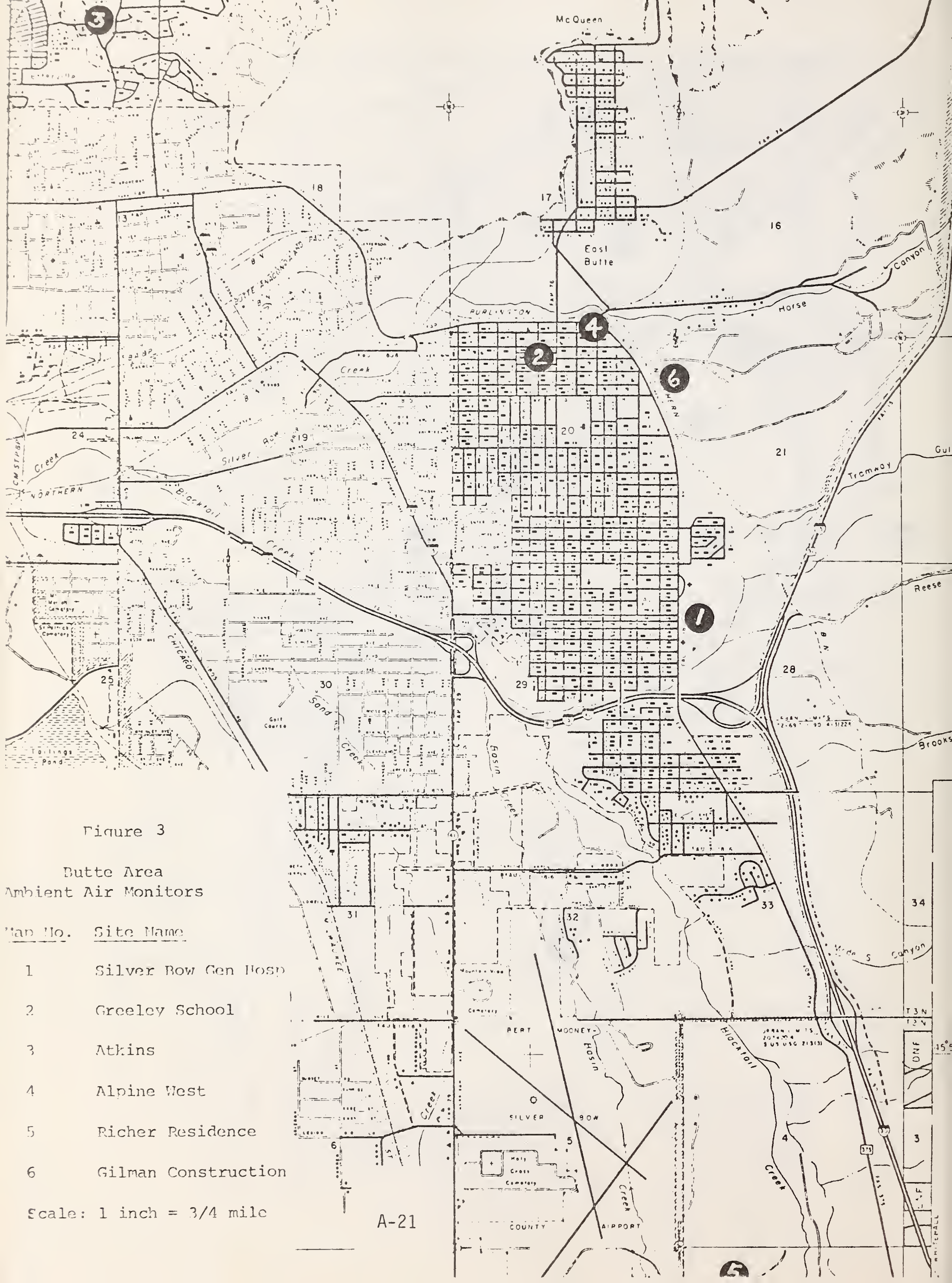

McQueen
East Butte
16
17
18
Burlington
Butte Anaconda and Pacific
Horse
Canyon
Creek
Northern
20
21
19
24
Silver Bow
Creek
Northern
Blacktail
Creek
Tramway
Gul
Reese
B N
28
29
30
25
Sand
Creek
Golf Course
Chicago
Tailings Pond
Basin
Creek
Brooks
31
32
33
34
Mountain View Cemetery
Basin
Creek
Blacktail
Creek
Silver Bow
County
Airport
Holy Cross Cemetery
Bert Mooney
Creek
T 3 N
T 2 N
DNF
6
5
4
3
1
2
3
4
5
6

Figure 3

Butte Area
Ambient Air Monitors

Map No.	Site Name
1	Silver Bow Gen Hosp
2	Greeley School
3	Atkins
4	Alpine West
5	Richer Residence
6	Gilman Construction

Scale: 1 inch = 3/4 mile

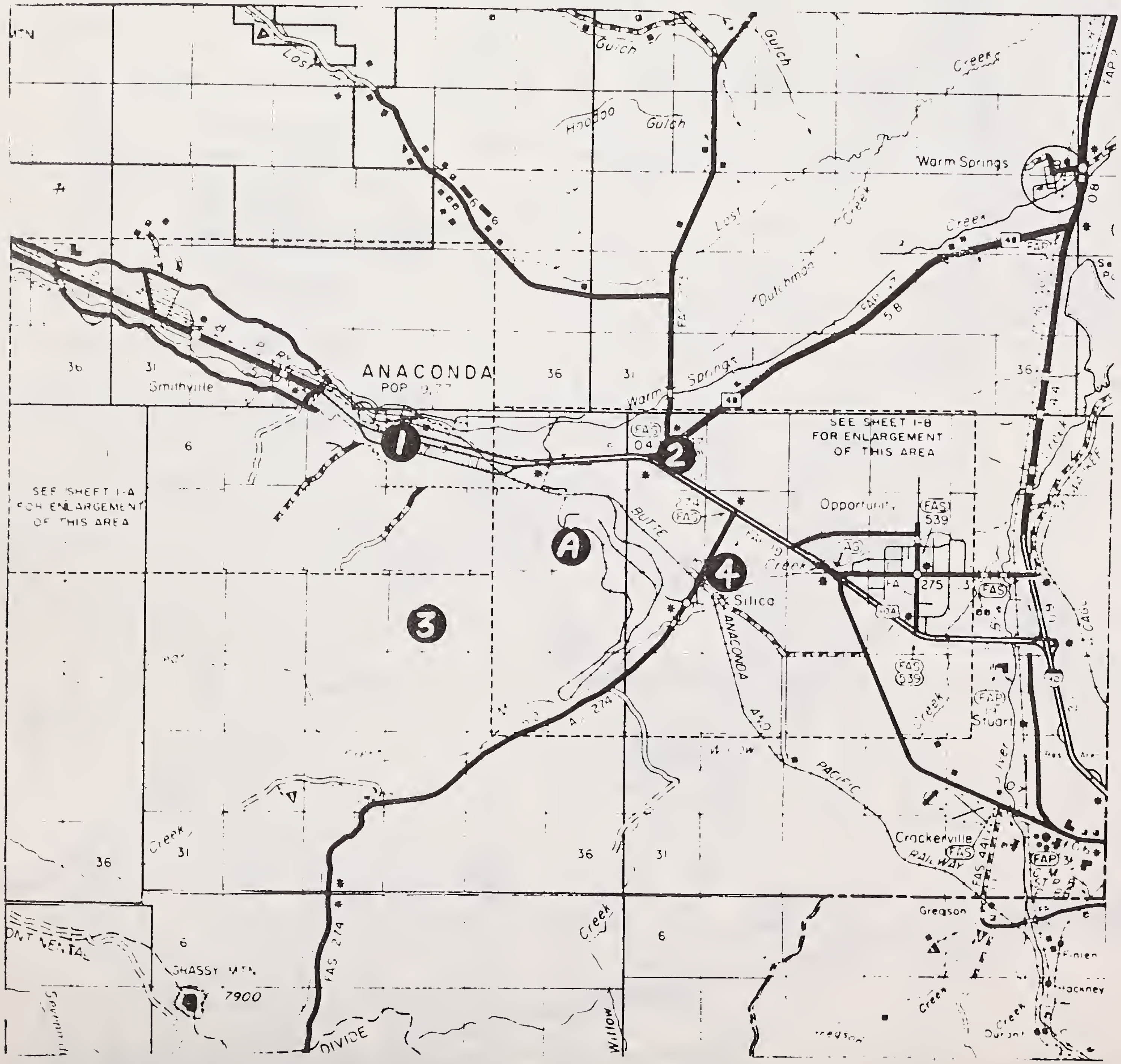

ANACONDA
POP 9,7
Smithville
SEE SHEET I-A FOR ENLARGEMENT OF THIS AREA
SEE SHEET I-B FOR ENLARGEMENT OF THIS AREA
1
2
3
4
A
Warm Springs
Opportunity
Silica
Crackerville
Gregson
Stuart
GRASSY MTN
7900
DIVIDE
BUTTE
ANACONDA
PACIFIC
RAILWAY
Hoodoo Gulch
Lost Creek
Dutchman Creek
Willow Creek
FAS 274
FAP 58
FAS 539
FAS 441
FAP 34
CONTINENTAL
Finlen
Durant

FIGURE 4

Anaconda Area
Ambient Air Monitors
and Major Point Soruce

Map No.	Site Name
1	Post Office
2	Hiway Junction
3	C-Hill
4	Mill Creek

Map No.	Source Name
A	Anaconda Company

Scale: 1 inch = 2 miles

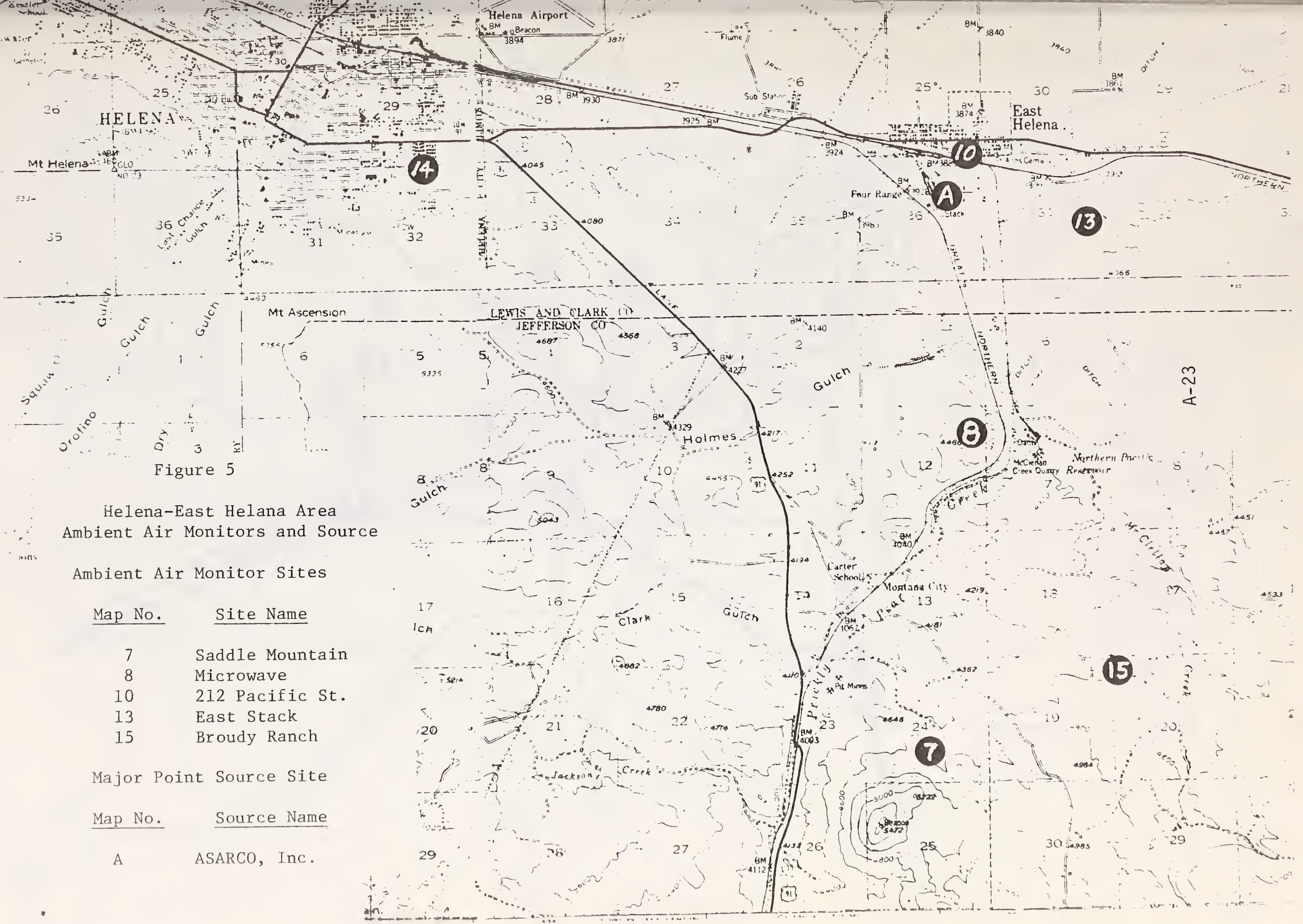

HELENA
Helena Airport
Mt Helena
Mt Ascension
LEWIS AND CLARK CO
JEFFERSON CO
HELENA CITY LIMITS
East Helena
Four Range
Stack
Holmes Gulch
Clark Gulch
Jackson Creek
Prickly Pear Creek
Carter School
Montana City
McClellan Creek Quarry
Northern Pacific Reservoir
Orofino Gulch
Dry Gulch
Last Chance Gulch
Squaw Gulch
GREAT NORTHERN
NORTHERN PACIFIC
Sub Station
Flume
14
10
A
13
8
15
7

Figure 5

Helena-East Helana Area
Ambient Air Monitors and Source

Ambient Air Monitor Sites

Map No.	Site Name
7	Saddle Mountain
8	Microwave
10	212 Pacific St.
13	East Stack
15	Broudy Ranch

Major Point Source Site

Map No.	Source Name
A	ASARCO, Inc.

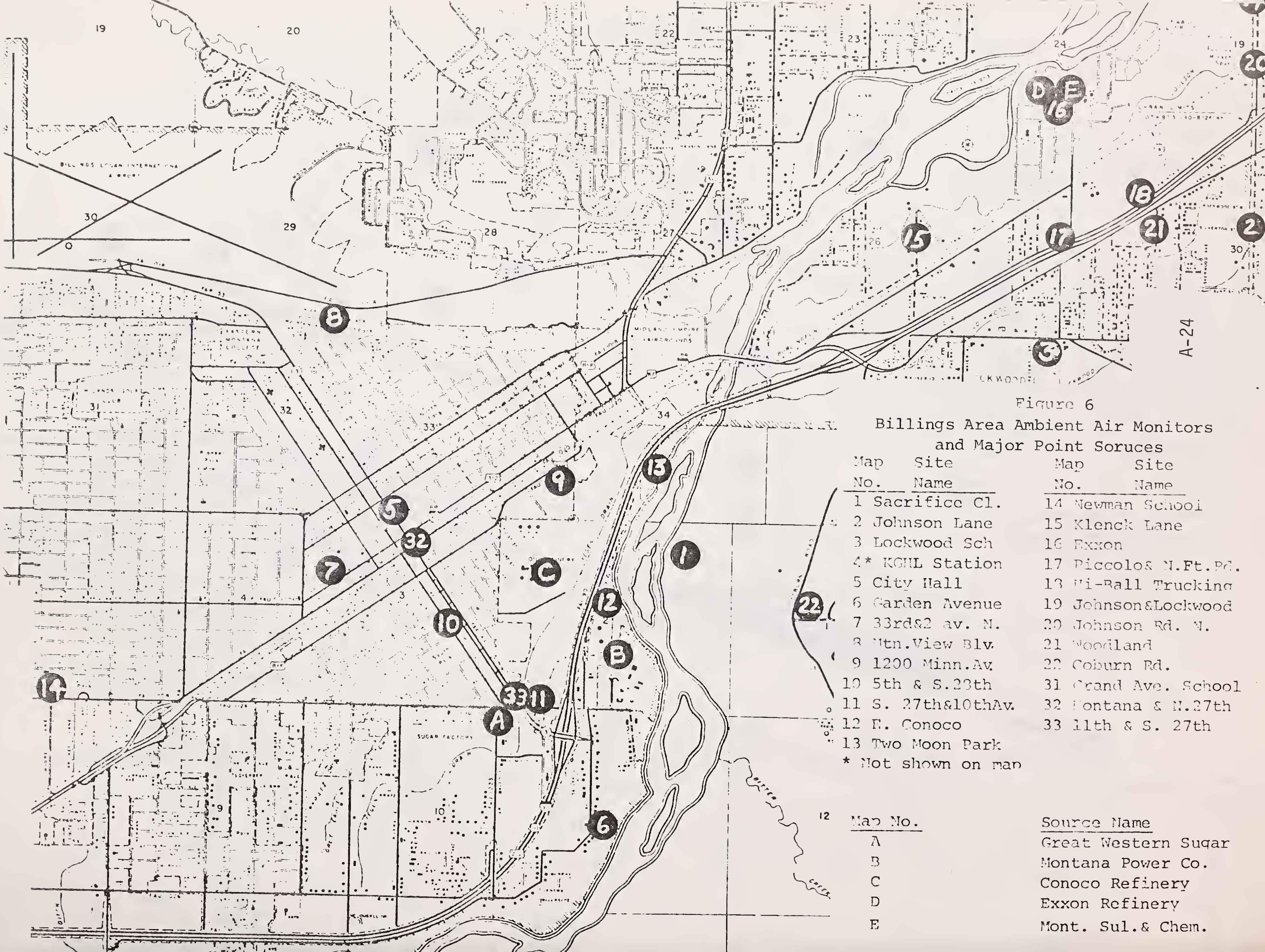

BILLINGS LOGAN INTERNATIONAL AIRPORT
EASTERN MONTANA COLLEGE
MIDLAND EMPIRE FAIRGROUNDS
SUGAR FACTORY
DRAIN
LOCKWOOD
1
2
3
5
6
7
8
9
10
11
12
13
14
15
16
17
18
20
21
22
32
33
A
B
C
D
E

Figure 6
Billings Area Ambient Air Monitors
and Major Point Soruces

Map No.	Site Name	Map No.	Site Name
1	Sacrifice Cl.	14	Newman School
2	Johnson Lane	15	Klenck Lane
3	Lockwood Sch	16	Exxon
4*	KGHL Station	17	Piccolo& N.Ft.Rd.
5	City Hall	18	Hi-Ball Trucking
6	Garden Avenue	19	Johnson&Lockwood
7	33rd&2 av. N.	20	Johnson Rd. N.
8	Mtn.View Blv.	21	Woodland
9	1200 Minn.Av.	22	Coburn Rd.
10	5th & S.28th	31	Grand Ave. School
11	S. 27th&10thAv.	32	Montana & N.27th
12	E. Conoco	33	11th & S. 27th
13	Two Moon Park		

* Not shown on map

Map No.	Source Name
A	Great Western Sugar
B	Montana Power Co.
C	Conoco Refinery
D	Exxon Refinery
E	Mont. Sul.& Chem.

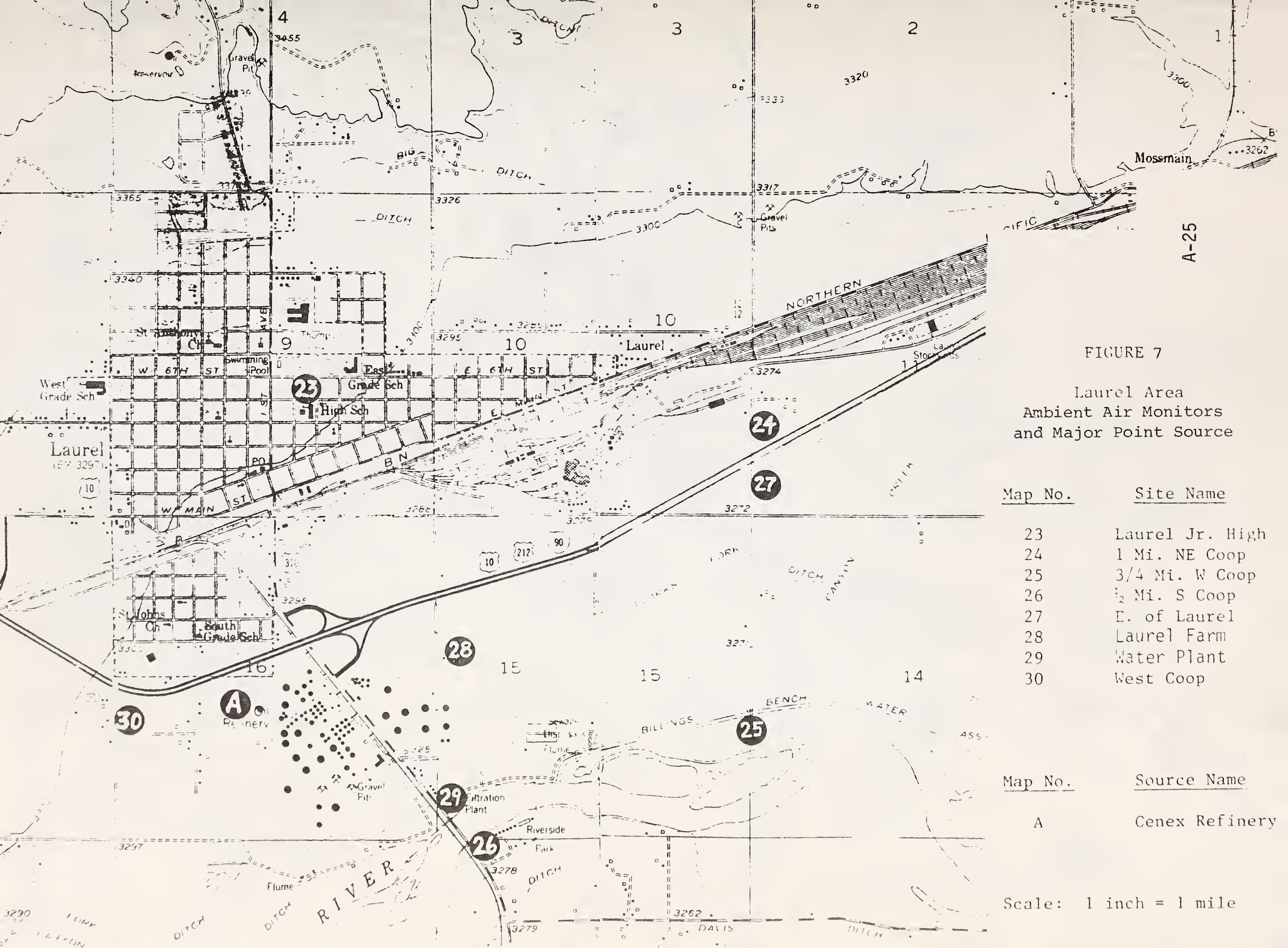

Laurel
West Grade Sch
St Anthony Ch
Swimming Pool
East Grade Sch
High Sch
W 6TH ST
E 6TH ST
W MAIN ST
E MAIN ST
AVE
PO
BN
NORTHERN
PACIFIC
St Johns Ch
South Grade Sch
Oil Refinery
Gravel Pit
Filtration Plant
Riverside Park
Flume
RIVER
DITCH
BIG DITCH
BILLINGS BENCH WATER ASS
Mossmain
Gravel Pits
Reservoir
10
212
90
23
24
25
26
27
28
29
30
A

FIGURE 7

Laurel Area
Ambient Air Monitors
and Major Point Source

Map No.	Site Name
23	Laurel Jr. High
24	1 Mi. NE Coop
25	3/4 Mi. W Coop
26	½ Mi. S Coop
27	E. of Laurel
28	Laurel Farm
29	Water Plant
30	West Coop

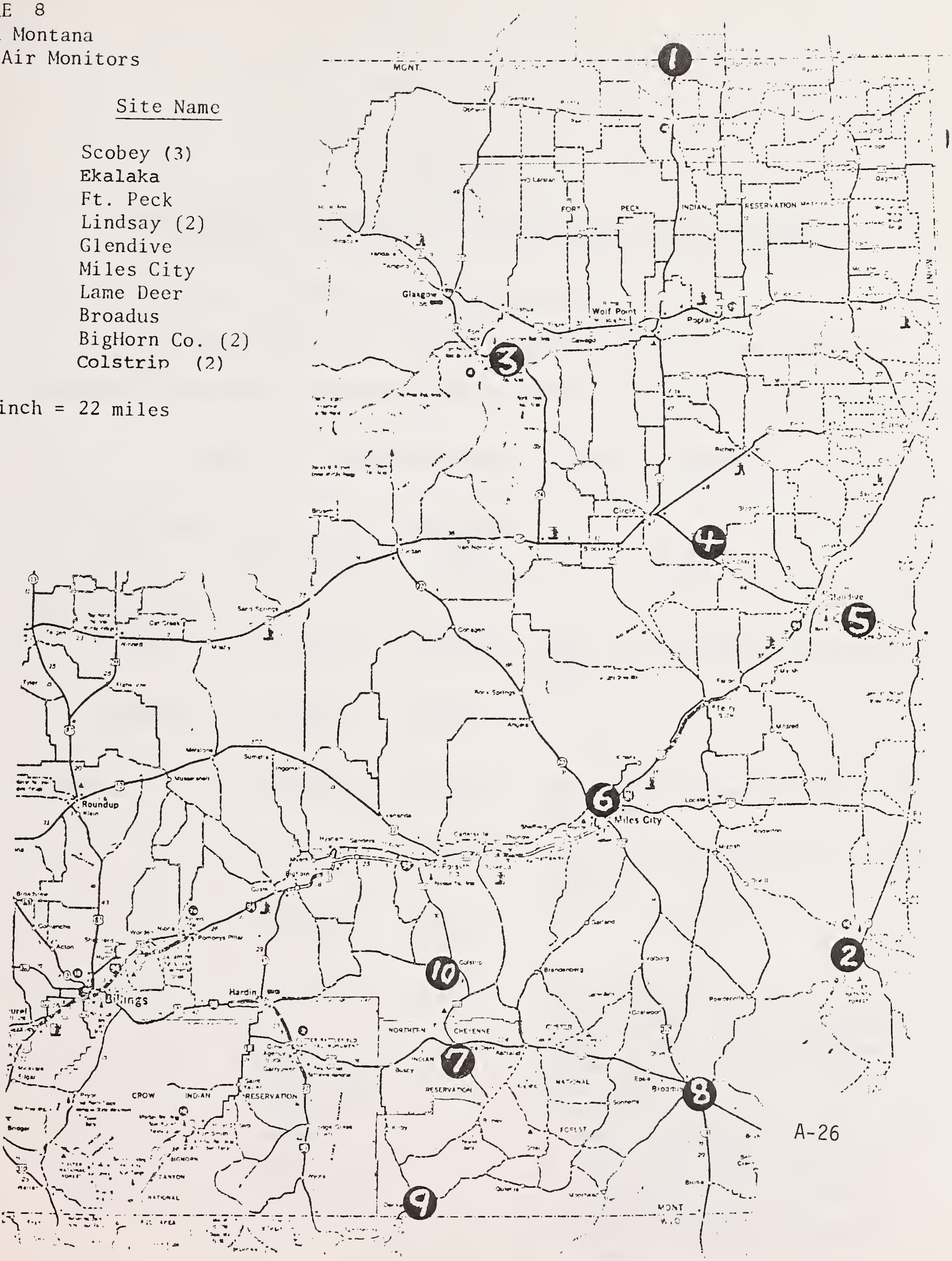
Map No.	Source Name
A	Cenex Refinery

Scale: 1 inch = 1 mile

FIGURE 8
Eastern Montana
Ambient Air Monitors

Map No.	Site Name
1	Scobey (3)
2	Ekalaka
3	Ft. Peck
4	Lindsay (2)
5	Glendive
6	Miles City
7	Lame Deer
8	Broadus
9	BigHorn Co. (2)
10	Colstrip (2)

ale: 1 inch = 22 miles


1
MONT.
FORT PECK INDIAN RESERVATION
Glasgow
Wolf Point
Poplar
3
Circle
4
Glendive
5
Roundup
Miles City
6
Billings
Hardin
10
NORTHERN CHEYENNE INDIAN RESERVATION
7
CROW INDIAN RESERVATION
CUSTER NATIONAL FOREST
Broadus
8
2
9
MONT
WYO

APPENDIX B.

AIR QUALITY STANDARDS OF OTHER STATES AND COUNTRIES

BI.I. AIR QUALITY STANDARDS IN THE UNITED STATES

The following tables list typical standards adopted by the federal government and several states for the pollutants discussed in this document.

TABLE B -I. SULFUR DIOXIDE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government			
Primary	0.14 ppm	24 hours	Not to be exceeded more than once per year.
Secondary	0.03 ppm	Annual	-
Alaska	0.50 ppm	3 hours	Not to be exceeded more than once per year.
	0.14 ppm	24 hours	Not to be exceeded more than once per year.
	0.03 ppm	Annual	-
California	0.5 ppm	1 hour	Alteration in lung function was found at this level in only one study. Other studies reported higher concentrations to cause this effect. a. Further studies on co-carcinogenic role are necessary. b. Does not include effects on vegetation, ecosystem and materials. c. May not include a margin of safety.
	0.05 ppm (When ozone and/or particulate exceed state standards)	24 hours	

Table B -I. (Cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
Florida	0.5 ppm	3 hours	Not to be exceeded more than once per year.
	0.1 ppm	24 hours	Not to be exceeded more than once per year.
	0.02 ppm	Annual	-
North Dakota	0.28 ppm	1 hour	-
	0.10 ppm	24 hours	-
	0.02 ppm	Annual	-
Pennsylvania	-	-	-
Wyoming	0.50 ppm	3 hours	Not to be exceeded more than once per year.
	0.10 ppm	24 hours	Not to be exceeded more than once per year.
	0.02 ppm	Annual	-

TABLE B -II SUSPENDED PARTICULATE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government Primary	260 ug/m^3	24 hours	Not to be exceeded more than once per year.
	75 ug/m^3	Annual	-
Secondary	150 ug/m^3	24 hours	Not to be exceeded more than once per year.
	60 ug/m^3	Annual	-

Table B -II (cont.)

Jurisdictional Area	Concentration	Averaging Time	Concentration
Alaska	150 ug/m^3	24 hours	Not to be exceeded more than once per year.
	60 ug/m^3	Annual	-
California	100 ug/m^3	24 hours	The standard applies to suspended particulate matter in general. It is not intended to be a standard for particles such as asbestos, lead, or berylium. Because size distribution influences the effect of particulate matter on health, the standard will be prescribed as data on health effects related to size distribution become available.
	60 ug/m^3	Annual	
Florida	150 ug/m^3	24 hours	Not to be exceeded more than once per year.
	60 ug/m^3	Annual	-
North Dakota	150 ug/m^3	24 hours	Not to be exceeded more than once per year.
	60 ug/m^3	Annual	-
Pennsylvania	-	-	-
Wyoming	150 ug/m^3	24 hours	Not to be exceeded more than once per year.
	60 ug/m^3	Annual	-

TABLE B -III SETTLED PARTICULATE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government Primary	-	-	-
Secondary	-	-	-
Alaska	-	-	-
California	-	-	-
Florida	-	-	-
North Dakota	$5.25g/m^2/mo.$	3 months	Residential areas
	$10.5g/m^2/mo.$	3 months	Heavy industrial areas
Pennsylvania	$15g/m^2/mo.$	30 days	-
	$8g/m^2/mo.$	Annual	-
Wyoming	$5g/m^2/mo.$	30 days	Residential areas. Includes $1.7g/m^2$ background.
	$10g/m^2/mo.$	30 days	

TABLE B -IV VISIBILITY AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government Primary	-	-	-
Secondary	-	-	-
Alaska	-	-	-
California	10 miles	one observation	When relative humidity is less than 70%.

Table B -IV (cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
California	30 miles	one observation	Lake Tahoe air basin. When relative humidity is less than 70%.
Florida	-	-	-
North Dakota	-	-	-
Pennsylvania	-	-	-
Wyoming	-	-	-

TABLE B -V FLOURIDE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government Primary	-	-	-
Secondary	-	-	-
Alaska	-	-	-
California	-	-	-
Florida	-	-	-
North Dakota	-	-	-
Pennsylvania	6.1 ppb	24 hours	Total soluble, as hydrogen fluoride.
Wyoming	1 ppb	24 hours	Measured as hydrogen fluoride. Or when the concentration of fluorides in forage for animal consumption, measured as fluorine, dry weight basis is 25 ppm.

Table B -V (cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
Wyoming	30 ug/cm^2	30 days	Measured by the sodium formate paper method or equivalent.

TABLE B -VI OZONE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government			
Primary	0.08 ppm	1 hour	Measured as ozone. Corrected for nitrogen dioxide and sulfur dioxide interference. Not to be exceeded more than once per year.
Secondary	0.08 ppm	1 hour	
Alaska	0.08 ppm	1 hour	Measured as ozone. Corrected for nitrogen dioxide and sulfur dioxide interference. Not to be exceeded more than once per year.
California	0.10 ppm	1 hour	Measured as ozone. This level is below that associated with aggravation of respiratory diseases.
Florida	0.08 ppm	1 hour	Measured as ozone. Corrected for nitrogen dioxide and sulfur dioxide interference. Not to be exceeded more than once per year.
North Dakota	0.08 ppm	1 hour	Measured as ozone. Corrected for nitrogen dioxide and sulfur dioxide interference. Not to be exceeded more than once per year.
Pennsylvania	-	-	-

Table B -VI (cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
Wyoming	0.08 ppm	1 hour	Measured as ozone. Corrected for nitrogen dioxide and sulfur dioxide interference. Not to be exceeded more than once per year.

TABLE B -VII NITROGEN DIOXIDE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government			
Primary	0.05 ppm	Annual	-
Secondary	0.05 ppm	Annual	-
Alaska	0.05 ppm	Annual	-
California	0.25 ppm	1 hour	-
Florida	0.05 ppm	Annual	-
North Dakota	0.10 ppm	1 hour	Not to be exceeded over one percent of the time in any three month period.
	0.05 ppm	Annual	-
Pennsylvania	-	-	-
Wyoming	0.05 ppm	Annual	-

TABLE B -VIII CARBON MONOXIDE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government Primary	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.
Secondary	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.
Alaska	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.
California	40 ppm	1 hour	This level is below those associated with impairment in time discrimination, visual function and psychomotor performance. At altitude lowered oxygen tension leads to greater absorption of carbon monoxide. Persons participating in strenuous recreational activities at higher altitudes are often unacclimated.
	6 ppm	8 hours	
	10 ppm	12 hours	
Florida	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.
North Dakota	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.

Table B -VIII (cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
Pennsylvania	-	-	-
Wyoming	35 ppm	1 hour	Not to be exceeded more than once per year.
	9 ppm	8 hours	Not to be exceeded more than once per year.

TABLE B -IX LEAD AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government	1.5 ug/m^3	90 days	-
Alaska	-	-	-
California	1.5 ug/m^3	30 days	-
Florida	-	-	-
North Dakota	-	-	-
Pennsylvania	5 ug/m^3	30 days	-
Wyoming	-	-	-

TABLE B -X HYDROGEN SULFIDE AIR QUALITY STANDARDS

Jurisdictional Area	Concentration	Averaging Time	Comments
Fed. Government	-	-	-

Table B -X (cont.)

Jurisdictional Area	Concentration	Averaging Time	Comments
Alaska	-	-	-
California	0.03 ppm	1 hour	-
Florida	-	-	-
North Dakota	0.032 ppm	½ hour	Not to be exceeded more than twice in any five consecutive days.
	0.054 ppm	½ hour	Not to be exceeded over twice per year.
Pennsylvania	0.1 ppm	1 hour	-
	0.005 ppm	24 hours	-
Wyoming	0.03 ppm	½ hour	Not to be exceeded more than two times in any consecutive five days.
	0.05 ppm	½ hour	Not to be exceeded more than two times per year.

BI.B. FOREIGN AIR QUALITY STANDARDS

Canada

The federal government of Canada has recognized three levels of air quality. The "Maximum Tolerable Level" is defined as the concentration sufficient to cause danger to public health and requiring immediate abatement action. The "Maximum Acceptable Level" is defined as the concentration that will provide adequate protection for health and the environment. When this level is exceeded it is necessary to reduce emissions. The "Maximum Desirable Level" defines a concentration below which there would be 'no effect' on any receptor. These federal standards are listed in

Table B -XI (Fisheries and Environment Canada, 1976, 1978). Provincial authorities may, and frequently do, adopt and enforce stricter standards.

TABLE B -XI CANADIAN FEDERAL AIR QUALITY STANDARDS

	Concentration	Averaging Time	Comments
Sulfur Dioxide	0.31 ppm	24 hours	Maximum Tolerable
	0.34 ppm	1 hour	Maximum Acceptable
	0.11 ppm	24 hours	Maximum Acceptable
	0.02 ppm	Annual	Maximum Acceptable
	0.17 ppm	1 hour	Maximum Desirable
	0.06 ppm	24 hours	Maximum Desirable
	0.01 ppm	Annual	Maximum Desirable
Suspended Particulate	400 ug/m^3	24 hours	Maximum Tolerable
	120 ug/m^3	24 hours	Maximum Acceptable
	70 ug/m^3	Annual	Maximum Acceptable
	60 ug/m^3	Annual	Maximum Desirable
Carbon Monoxide	17 ppm	8 hours	Maximum Tolerable
	30 ppm	1 hour	Maximum Acceptable
	13 ppm	8 hours	Maximum Acceptable
	13 ppm	1 hour	Maximum Desirable
	5 ppm	8 hours	Maximum Desirable
Ozone	0.15 ppm	1 hour	Maximum Tolerable
	0.08 ppm	1 hour	Maximum Acceptable
	0.025 ppm	24 hours	Maximum Acceptable
	0.015	Annual	Maximum Acceptable

Table B -XI (cont.)

	Concentration	Averaging Time	Comments
Ozone (cont.)	0.05 ppm	1 hour	Maximum Desirable
	0.015 ppm	24 hours	Maximum Desirable
	0.010 ppm	Annual	Maximum Desirable
Nitrogen Dioxide	0.53 ppm	1 hour	Maximum Tolerable
	0.16 ppm	24 hours	Maximum Tolerable
	0.21 ppm	1 hour	Maximum Acceptable
	0.11 ppm	24 hours	Maximum Acceptable
	0.05 ppm	Annual	Maximum Acceptable
	0.03 ppm	Annual	Maximum Desirable

The standards adopted by Saskatchewan are an example of provincial ambient air quality standards. They are listed in Table B -XII (Environment Saskatchewan, 1975).

TABLE B -XII SASKATCHEWAN AIR QUALITY STANDARDS

	Concentration	Averaging Time	Comments
Sulfur Dioxide	0.17 ppm	1 hour	-
	0.06 ppm	24 hours	-
	0.01 ppm	Annual	-
Sulfation	0.3 mg/cm^2	30 days	-
Suspended Particulate	120 ug/m^3	24 hours	-
	70 ug/m^3	Annual	-
Settled Particulate	20 g/m^2	30 days	-

Table B -XII (cont.)

	Concentration	Averaging Time	Comments
Soiling Index	1.5 CoH	24 hours	-
Carbon Monoxide	13 ppm	1 hour	-
	5 ppm	8 hours	-
Ozone	0.05 ppm	1 hour	-
	0.015 ppm	24 hours	-
Nitrogen Dioxide	0.2 ppm	1 hour	-
	0.1 ppm	24 hours	-
	0.05 ppm	Annual	-

West Germany

No permit can be granted for a new industrial facility that would result in an ambient standard being exceeded. In areas where the ambient standards are already exceeded, industries must meet detailed emission limits and construct new stacks to raise the point of emissions. The West German Ambient Standards are listed in Table AI-XIII (Budesminister des Innern, 1974).

TABLE B -XIII WEST GERMAN AIR QUALITY STANDARDS

	Concentration	Averaging Time	Comments
Sulfur Dioxide	0.14 ppm	short-term	-
	0.05 ppm	long-term	-
Suspended Particulate	400 ug/m^3	short-term	-
	200 ug/m^3	long-term	-

Table B -XIII (cont.)

	Concentration	Averaging Time	Comments
Fine Particulate	200 ug/m^3	short-term	Less than 10 um
	100 ug/m^3	long-term	Less than 10 um
Settled Particulate	20 g/m^2/mo.	short-term	-
	10 g/m^2/mo.	long-term	-
Fluoride	4.5 ppb	short-term	-
	2.0 ppb	long-term	-
Carbon Monoxide	24 ppm	short-term	-
	8 ppm	long-term	-
Nitrogen Dioxide	0.15 ppm	short-term	-
	0.05 ppm	long-term	-
Hydrogen Sulfide	7 ppb	short-term	-
	3 ppb	long-term	-

Japan

In areas where the ambient air quality standards are exceeded the prefectural authorities must devise a control plan which will achieve the standards in five years. The consideration of stack height and dispersion as a control technique for sulfur dioxide was removed from the Law in 1974. New plants cannot be granted a construction permit in areas where ambient standards are exceeded or if their emissions would cause the ambient standards to be exceeded. Where air pollution becomes serious, the central government assumes responsibility for plans and regulations. The Japanese ambient air quality standards are listed in Table AI-XIV (Environment Agency, Japan, 1976).

TABLE B -XIV AIR QUALITY STANDARDS OF JAPAN

	Concentration	Averaging Time	Comments
Sulfur Dioxide	0.1 ppm	1 hour	-
	0.04 ppm	24 hours	-
Fine Particulate	200 ug/m^3	1 hour	Less than 10 um
	100 ug/m^3	24 hours	Less than 10 um
Carbon Monoxide	20 ppm	8 hours	-
	10 ppm	24 hours	-
Ozone	0.06 ppm	1 hour	-
Nitrogen Dioxide	0.02 ppm	24 hours	-

Other Countries

Information is not readily available on the legal role of air quality standards in all countries. In many countries the primary emphasis is on emission limits, with air quality standards entirely as guidelines. In other countries, their legal role is similar to that in the United States, West Germany, and Japan. Standards for a selection of countries typical of different philosophies and political systems are listed in Tables AI-XV through AI-XXII (Jarrault, 1976).

TABLE B -XV FOREIGN SULFUR DIOXIDE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
France	0.26 ppm	24 hours	Not to be exceeded more than eight days per year.

Table B -XV (cont.)

Country	Concentration	Averaging Time	Comments
Holland	0.03 ppm	Annual	In presence of particulate
	0.09 ppm	24 hours	In the presence of particulate. Not to be exceeded more than seven days a year.
	0.05 ppm	Annual	When particulate is less than 30 ug/m^3.
	0.12 ppm	24 hours	When particulate is less than 90 ug/m^3. Not to be exceeded more than seven days a year.
Israel	0.3 ppm	30 minutes	Not to be exceeded more than four days a year.
	0.10 ppm	24 hours	Not to be exceeded more than four days a pear.
Sweden	0.24 ppm	30 minutes	-
	0.10 ppm	24 hours	-
	0.05 ppm	30 days	-
U.S.S.R.	0.17 ppm	30 minutes	Not to be exceeded
	0.02 ppm	24 hours	Not to be exceeded

TABLE B -XVI FOREIGN SUSPENDED PARTICULATE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
France	350 ug/m^3	24 hours	Not to be exceeded more than eight days a year.
Israel	200 ug/m^3	24 hours	Not to be exceeded more than eight days a year.
	75 ug/m^3	Annual	-

Table B -XVI (cont.)

Country	Concentration	Averaging Time	Comments
Sweden	100 ug/m^3	1 hour	Not to be exceeded more than eight hours in one month.
U.S.S.R.	500 ug/m^3	30 minutes	Non-toxic, inert particles
	150 ug/m^3	24 hours	Non-toxic, inert particles
	150 ug/m^3	30 minutes	Chimney soot
	50 mg/m^3	24 hours	Chimney soot

TABLE B -XVII FOREIGN FLUORIDE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Israel	3.4 ppb	30 minutes	Total fluoride not to be exceeded more than four days a year.
	1.1 ppb	24 hours	Total fluoride not to be exceeded more than four days a year.
Spain	3.4 ppb	30 minutes	Gaseous fluoride only
	1.1 ppb	24 hours	Gaseous fluoride only
	7 ppb	30 minutes	Total fluoride
	2 ppb	24 hours	Total fluoride
U.S.S.R.	2.3 ppb	20 minutes	Gaseous fluoride only
	0.6 ppb	24 hours	Gaseous fluoride only
	3.4 ppb	20 minutes	Total fluoride
	1.1 ppb	24 hours	Total fluoride

TABLE B -XVIII FOREIGN OZONE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Israel	0.20	30 minutes	Not to be exceeded more than four days a year.
	0.10	8 hours	Not to be exceeded more than four days a year.
Romania	0.02	24 hours	

TABLE B -XIX FOREIGN NITROGEN DIOXIDE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Finland	0.3 ppm	30 minutes	-
	0.11 ppm	24 hours	-
Spain	0.2 ppm	30 minutes	-
	0.1 ppm	24 hours	-
	0.05 ppm	Annual	-
Romania	0.15 ppm	30 minutes	-
	0.05 ppm	24 hours	-
U.S.S.R.	0.045 ppm	30 minutes	-

TABLE B -XX FOREIGN CARBON MONOXIDE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Israel	30 ppm	30 minutes	Not to be exceeded more than four days a year.

Table B -XX (cont.)

Country	Concentration	Averaging Time	Comments
Israel	10 ppm	8 hours	Not to be exceeded more than four days a year.
Italy	50 ppm	30 minutes	Not to be exceeded more than once in eight hours.
	20 ppm	8 hours	-
Spain	35 ppm	30 minutes	-
	12 ppm	8 hours	-
U.S.S.R.	2.4 ppm	30 minutes	-
	0.8 ppm	24 hours	-

TABLE B -XXI FOREIGN LEAD AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Israel	5 ug/m^3	24 hours	Not to be exceeded more than four days a year.
Italy	50 ug/m^3	30 minutes	Not to be exceeded more than once in eight hours.
	10 ug/m^3	24 hours	-
Romania	1 ug/m^3	24 hours	-
U.S.S.R.	0.7 ug/m^3	24 hours	-

TABLE B. -XXII FOREIGN HYDROGEN SULFIDE AIR QUALITY STANDARDS

Country	Concentration	Averaging Time	Comments
Israel	0.1 ppm	30 minutes	-
	0.03 ppm	24 hours	-
Italy	0.07 ppm	30 minutes	Not to be exceeded more than once in eight hours.
	0.03 ppm	24 hours	-
Romania	0.02 ppm	30 minutes	-
	0.10 ppm	24 hours	-
U.S.S.R.	0.005 ppm	30 minutes	-

Literature Cited

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APPENDIX C

PROPOSED AMBIENT AIR QUALITY STANDARDS RULE

This rule is proposed to replace the existing ambient air quality standards rule 16-2.14(1)-S14040 of the Administrative Rules of Montana, which is reproduced in Appendix D. The revision to the existing rule is being proposed to ensure the enforceability of the standards and to reflect the latest scientific information on the effects of air pollution. Other changes have also been made in the rule to clarify their intent and applicability. The following existing air quality standards are being deleted by this proposal: sulfation, suspended sulfate, sulfuric acid mist, and beryllium. This is due primarily to difficulties in measuring the pollutants or a lack of scientific information on which to base a standard. The department will continue to review developments in measurement techniques and scientific knowledge with the intent of proposing ambient air quality standards for the foregoing four pollutants at such time as adequate information is available. The department is also initiating a review of the scientific information on acid rain, fine particles, cadmium, arsenic and carcinogenic organic compounds with the intent of proposing ambient air quality standards for them whenver adequate information is available. New air quality standards are being added by this rule relating to visibility, ozone, nitrogen dioxide and carbon monoxide. Other changes include a definition of "ambient air", a new definition of standard atmospheric conditions, a clear statement of the requirements for data to be considered

valid, and the substitution of a new statement of purpose for the ambient air quality standards.

16-2.14(1)-S14040 AMBIENT AIR QUALITY STANDARDS

(1) Purpose

(a) It is the primary purpose of these ambient air quality standards to establish maximum levels of air contaminants that, given present knowledge, will not adversely affect the health or safety of persons. A margin of safety is incorporated to insure that human health and safety are protected. In some instances standards have been adopted that are more stringent than would be necessary only to protect the public health. This is necessary to prevent injury to plant and animal life and property, foster the comfort and convenience of the people, promote the economic and social development of this state and facilitate the enjoyment of the natural attractions of this state, to the greatest degree practicable.

(b) These standards are legally enforceable limitations and any source causing or contributing to a violation of this rule is subject to enforcement proceedings. These standards are to be achieved primarily by the reduction of emissions of air contaminants to the outdoor atmosphere by the enforcement of rules on the emissions of air contaminants passed pursuant to the Montana Clean Air Act. Compliance with all emissions limitations does not relieve any source from the need to insure compliance with the ambient air quality standards.

(2) Definitions:

"Air quality standard" means a maximum permissible level of an air contaminant in the ambient air as defined by the maximum frequency with which a specified level may be exceeded or by a maximum level of an air contaminant in body or plant tissues.

"Ambient air" means that part of the outdoor atmosphere excluding the space encompassing and immediately adjacent to the structures and activity area of air pollution sources. If, however, the public has access to any portion of the atmosphere exluded by the previous sentence, that portion of the outdoor atmosphere is ambient air.

"Annual average" means an arithmetic average of all valid recorded values during any twelve (12) consecutive months, provided that:

(i) at least forty (40) 24-hour averages valid recorded values are necessary and each of these values must be separated from the previous value by at least six (6) days, or

(ii) at least 5840 hourly average valid recorded values are necessary.

"Calendar quarter average" means an arithmetic average of all valid recorded values during the months of January through March, April through June, July through September, or October through December. The minimum number of valid recorded values shall be ten (10), provided that each of these values must be separated from the previous value by at least six (6) days.

"Carbon monoxide" means the gas having the molecular composition of one carbon and one oxygen atom.

"Eight (8) hour average" means the arithmetic average of all valid recorded values during any consecutive eight (8) hours, but not less than six (6) non-overlapping valid hourly averages.

"Equivalent method" means any method of sampling and analyzing for an air contaminant that has been demonstrated to the satisfaction of the department to be equivalent in sensitivity, accuracy, reproducibility and selectivity to the sampling methods specified in this rule at or near the level of the air quality standard.

"Fluoride" means the gas having the molecular composition of one fluorine atom and one hydrogen atom.

"Foliar fluoride" means fluorine combined with other substances in or on vegetation.

"Forage" means any plant part which is grazed or browsed at that location.

"Growing season average" means an arithmetic average of all valid recorded values during the period between April 1 and September 1, inclusive, of any year, but not less than one hundred two (102) twenty-four (24) hour average valid recorded values.

"Grams per square meter" (abbreviated gm/m^2) means a concentration numerically equal to the mass of an air contaminant (in grams) deposited on one square meter of surface.

"Hourly average" means an arithmetic average of all valid values recorded between the first minute and the sixtieth minute of the hour (e.g., 1:00 to 2:00), but not less than two-thirds (2/3) of the data obtainable from the monitoring device during the hour, or an integral sample of more than forty (40) minutes.

"Hydrogen sulfide" means the gas having the molecular composition of one sulfur atom and two hydrogen atoms.

"Lead" means elemental lead or lead in combination with any other substance.

"Micrograms per cubic meter" (abbreviated ug/m^3) means a concentration numerically equal to the mass of an air contaminant present (in micrograms) in a one cubic meter of air, corrected to standard conditions.

"Micrograms per gram" (abbreviated ug/g) means a concentration numerically equal to the mass of an air contaminant (in micrograms) in one gram of dry material.

"Nitrogen dioxide" means the gas having the molecular composition of one nitrogen atom and two oxygen atoms.

"Outdoor atmosphere" means that portion of the atmosphere which is external to any building or structure.

"Ozone" means the gas having the molecular composition of three oxygen atoms.

"Particle scattering coefficient" means the light scattering power per meter of airborne particulate matter.

"Parts per million" (abbreviated ppm) means a concentration of an air contaminant numerically equal to the volume of a gaseous air contaminant present in one million volumes of air at the same conditions of temperature and pressure.

"Parts per billion" (abbreviated ppb) means a concentration of an air contaminant numerically equal to the volume of a gaseous air contaminant present in one billion volumes of air at the same conditions of temperature and pressure.

"Standard conditions" means a temperature of 25^{0} Celsius and a pressure of 760 millimeters of mercury.

"Sulfur dioxide" means the gas having the molecular composition of one sufur atom and two oxygen atoms.

"Thirty (30) day average" means an arithmetic average of all valid recorded values during any consecutive thirty (30) days, but not less than twenty (20) valid twenty-four hour average recorded values or an integral sample of more than twenty (20) days.

"Twenty-four (24) hour average" means an arithmetic average of each valid recorded value during any consecutive twenty-four (24) hours, but not less than eighteen (18) valid hourly averages or an integral sample of more than eighteen (18) hours.

"Valid recorded value" means data recorded, collected, transmitted and analyzed as required by Section 4 of this rule.

"Year" means any twelve (12) consecutive months.

(3) Ambient Air Quality Standards

No person shall cause or authorize the emission of any air contaminant from a source, alone or in combination with the eimssions from other sources, such that the concentration in the ambient air exceeds any air quality standard established in this rule. The following air quality standards are maximum values that may not be exceeded:

(a) Carbon monoxide:

  (i) 9 ppm 8-hour average, not to be exceeded more than one a year;

  (ii) 17 ppm hourly average, not to be exceeded more than once a year;

  (iii) Concentrations of carbon monoxide shall be measured by the non-dispersive infrared method, as more fully described in the Code of Federal Regulations (1978 compilation), Title 40, Part 50, Appendix C, or by an approved equivalent method.

(b) Foliar fluoride:

  (i) 30 ug/g;

  (ii) Concentrations of foliar fluoride shall be determined from forage collected according to the procedures for sampling vegetation in Air Quality Methods and Procedures (1978), Air Quality Bureau, Montana Department of Health and Environmental Sciences, Section No. 1.6.7.1, and analysed by the semi-automated method, as more fully described in Methods of Air Sampling and Analysis, Second Edition (1977), Method No. 12202-02-68T, provided that the surfaces of the plant material are not to be washed, or by an approved equivalent method.

(c) Fluoride:

  (i) 1.0 ppb 24-hour average;

  (ii) 0.30 ppb 30-day average;

  (iii) 0.13 ppb growing season average;

  (iv) Concentrations of fluoride shall be measured by the double tape sampler method, as more fully described in Methods of Air Sampling and Analysis, Second Edition (1977) Method No. 42222-02-72T, as modified by the addition of a heated stainless steel sample inlet line, with the NaOH-impregnated tape analysed by the semiautomated method described in Methods of Air Sampling and Analysis, Second Edition (1977) Method 12202-02-68T, Section 7.3, or by an approved equivalent method.

(d) Hydrogen Sulfide:

  (i) .10 ppm hourly average;

(ii) Concentrations of hydrogen sulfide shall be measured by the methylene blue spectrophotometric method, as more fully described in Methods of Air Sampling and Analysis, Second Edition (1977), Method P & CAM 126-C, or by an approved equivalent method.

(e) Lead:

(i) 1.5 ug/m^3 calender quarter average;

(ii) Concentrations of lead shall be measured by the atomic absorption method, as more fully described in the Federal Register (October 5, 1978), pp. 46258-61, or by an approved equivalent method.

(f) Nitrogen dioxide:

(i) 0.05 ppm annual average;

(ii) 0.17 ppm hourly average, not to be exceeded more than once a year;

(iii) Concentrations of nitrogen dioxide shall be measured by the chemiluminescence method, as more fully described in the Code of Federal Regulations (1978 compilation), Title 40, Part 50, Appendix F, or by an approved equivalent method.

(g) Ozone:

(i) 0.10 ppm hourly average; not to be exceeded more than once a year;

(ii) Concentrations of ozone shall be measured by the chemiluminescence method, as more fully described in the Code of Federal Regulations (1978 compilation), Title 40, Part 50, Appendix D, or by an approved equivalent method.

(h) Settled particulate matter:

  (i) 10 gm/m^2 30-day average;

  (ii) Concentration of settled particulate shall be measured by the dustfall method, as more fully described in _Methods of Air Sampling and Analysis, Second Edition_ (1977), Method No. 21101-0170T, or by an approved equivalent method.

(i) Sulfur dioxide:

  (i) 0.02 ppm annual average;

  (ii) 0.10 ppm 24-hour average not to be exceeded more than once a year;

  (iii) 0.40 ppm hourly average not to be exceeded more than once a year;

  (iv) Concentrations of sulfur dioxide shall be measured by the pararosaniline method as more fully described in the Code of Federal Regulations (1978 compilation) Title 40, Part 50, Appendix A, or by an approved equivalent method.

(j) Suspended particulate matter:

  (i) 75 ug/m^3 annual average;

  (ii) 200 ug/m^3 24-hour average, not to be exceeded more than once a year;

  (iii) Concentrations of suspended particulate shall be measured by the high volume method as more fully described in the Code of Federal Regulations (1978 compilation) Title 40, Part 50, Appendix B, or by an approved equivalent method.

(k) Visibility:

  (i) Particle scattering coefficient of 2 x 10^{-5} per meter annual average;

(ii) Visibility shall be measured by the integrating nephelometer method, as more fully described in Methods of Air Sampling and Analysis, Second Edition (1977), Method No. 11203-03-76T, or by an approved equivalent method.

(4) Valid Data

All sampling and data collection, recordings, analysis, and transmittal, including but not limited to site selection, calibrations, precision and accuracy determinations, and audits, shall be performed as specified in Appendices A through E, Federal Register, vol. 43, pp. 35906-35928, August 7, 1978.

(5) Enforcement

This rule is enforceable against any source causing or contributing to air pollution which exceeds the air quality standards established in this rule. Any source in violation of this rule is subject to the remedies described in the Clean Air Act.

APPENDIX D

EXISTING AMBIENT AIR QUALITY STANDARDS RULE

16-2.14(1)-S14040 AMBIENT AIR QUALITY STANDARDS

(1) In accordance with Section 69-3909, subsection 12 of the Clean Air Act of Montana, on May 7, 1967, the board adopted the Ambient Air Quality Standards shown below.

(2) Until additional pertinent information becomes available with respect to the effects of the substances listed below, the following air quality criteria shall apply in Montana.

Pollutants	Standards (Maximum permissible concentrations)
Sulfur Dioxide[a]	0.02 ppm, maximum annual average 0.10 ppm, 24-hour average, not to be exceeded over 1 percent of the days in any 3-month period 0.25 ppm not to be exceeded for more than one hour in any 4 consecutive days
Reactive sulfur (sulfation[b])	0.25 milligrams sulfur trioxide per 100 square centimeters per day, maximum annual average 0.50 milligram sulfur trioxide per 100 square centimeters per day, maximum for any 1-month period
Suspended sulfate[c]	4 micrograms per cubic meter of air, maximum allowable annual average 12 micrograms per cubic meter of air, not to be exceeded over 1 percent of the time
Sulfuric acid mist[d]	4 micrograms per cubic meter of air, maximum allowable annual average 12 micrograms per cubic meter of air, not to be exceeded over 1 percent of the time 30 micrograms per cubic meter of air, hourly average, not to be exceeded over 1 percent of the time
Hydrogen sulfide[e]	0.03 ppm, ½ hour average, not to be exceeded more than twice in any 5 consecutive days 0.05 ppm, ½ hour average, not to be exceeded over twice a year

Pollutants	Standards (Maximum permissible concentrations)
Total suspended particulate[f]	75 micrograms per cubic meter of air, annual geometric mean 200 micrograms per cubic meter of air, not to be exceeded more than 1 percent of days a year
Settled particulate (dustfall[g])	15 tons per square mile per month, 3-month average in residential areas 30 tons per square mile per month, 3-month average in heavy industrial areas
Lead[h]	5.0 micrograms per cubic meter of air, 30-day average
Beryllium[i]	0.01 micrograms per cubic meter of air, 30-day average
Fluorides, total (as HF) in air[j]	1 part per billion parts of air, 24-hour average
Fluorides (as F) in forage[k] for animal consumption - dry weight basis	35 parts per million
Fluorides (gaseous)[l]	0.3 micrograms per square centimeter per 28 days

a. Sulfur dioxide measured by West-Gaeke or conductometric method.

b. Sulfation measured by lead-peroxide candle.

c. Suspended sulfate measured by high-volume sampler - turbidmetric procedure.

d. Sulfuric acid mist - Air Pollution Control District, County of Los Angeles, APCD - Sulfuric acid 13-49.

e. Hydrogen sulfide measured by methylene blue method - acetate tape for screening and monitoring.

f. Suspended particulate measured by high-volume sampler.

g. Dustfall measured by container open to atmosphere.

h. Suspended lead measured by high-volume sampler - dithizone and/or spectrophotometric procedures.

i. Suspended beryllium measured by high-volume sampler - fluorometric and/or spectrophotometric method.

j. Total fluoride measured by impingers - Winter-Willard distillation procedure (SPADNS color).

k. Forage cut, dried, ashed and subjected to Winter-Willard distillation procedure (SPADNS color).

l. Gaseous fluorides measured by calcium formate paper technique in board standard shelter - Winter-Willard distillation procedure (SPADNS color).

The ambient air quality standards listed describe a level of air quality designed to protect people from the adverse effects of air pollution; and they are intended further to promote maximum comfort and enjoyment in use of property consistent with economic and social well-being of the community.

Ambient air quality standards are used as a tool in achieving cleaner air, not as a license to permit unnecessary degradation of air quality which would thwart attainment of the long-range goal to maintain a reasonable degree of air purity.

These standards are not intended to represent the ultimate in air quality achievement. It is anticipated that research and development will gradually make possible cleaner air at lower cost. As evidence accumulates on deleterious effect of the contaminant, present objectives will be revised or additional standards established. The standards are designed to protect the health, welfare and comfort of the public and to minimize economic losses.

Because some pollutants combine chemically to form more harmful materials than the original emissions, ascribing a single effect to a single pollutant would be an erroneous over-simplification. The standards, therefore, apply to air containing a variety of pollutants. Although reaching the goals will result in benefits, no allowance for the time needed to achieve them was considered in their selection. They are intended to apply to areas where people live or where an adverse effect may occur.

The board, in adopting these standards, intends them to be goals and guidelines and so interprets the legislative intent of the word "standards" in Section 69-3909 of the Clean Air Act of Montana.

(3) The sampling and analytical procedures employed to measure ambient levels of contaminants are to be consistent with obtaining accurate results

which are representative of the conditions being evaluated. The sampling and analytical techniques enumerated may be used directly or employed as reference standards against which other methods may be calibrated.

APPENDIX E
PROPOSED MEASUREMENT METHODS

The following measurement methods are to be used in determining the concentrations of regulated pollutants in the ambient air. In addition to these methods, others may be used if they are found by the Department to be equivalent to these methods in sensitivity, accuracy, reproducibility and selectivity at or near the concentration of the ambient air quality standard.

Carbon Monoxide

Carbon monoxide is measured by the non-dispersive infrared (IR) spectrometry method adopted by EPA under the federal Clean Air Act (40 CFR Part 50, App. C). This method is based on the absorption of infrared radiation by carbon monoxide in a non-dispersive photometer. Two infrared light beams in the photometer pass into matched cells, one containing a standard atmosphere and the other containing the sample to be measured. The carbon monoxide in the cell absorbs infrared radiation only at its characteristic frequencies. The detectors are sensitive to those frequencies. With a non-absorbing gas in the standard cell, and no carbon monoxide in the sample cell, the signals from both detectors are electronically balanced. Any carbon monoxide introduced into the sample cell will absorb radiation, which reduces the radiation received by the detector. This change is detected electronically and amplified to provide an output signal which can be interpreted in ppm s of carbon monoxide in the air.

Foliar Fluoride

For most pollutants the collection of a sample of the pollutant is a standard procedure and does not require any detailed explanation. Because standard techniques for the collection of plant materials for fluoride content measurement have not been developed, a detailed methodology is set out here.

In order to reduce variability in sampling, the following procedures must be followed:

1. Conduct a brief field reconnaissance of the sampling area to determine which plant species are being grazed. Herbivorous animals' grazing patterns and preferences for various plant species vary with season, location, grazing pressure and other variables, so it is necessary to visit each area to be sampled. The field reconnaissance should consist of walking through the area and recording which plant species show grazing use.

2. Locate sample locations accurately on U. S. Geological Survey Quadrangle maps when these are available or on aerial photographs, so the sample site can be relocated accurately. Verbal descriptions are adequate if the area can be defined easily.

3. Determine the genus and species of the plants being sampled.

4. Record the date and time of sampling.

5. Collect at least 200 grams of plant tissue from the grazeable portions of plants in a given location. If grasses are being sampled, the aerial portion of the plant should be collected, including old and new leaves. If shrubs or trees are being sampled, record which years of growth are being sampled. On trees which retain the needles or leaves for more than one year this is important since the older leaves may have considerably higher fluoride levels than the younger. For trees or shrubs which drop their leaves each year it can be assumed that the leaves are roughly the same age. In all cases entire leaves should be collected and analyzed rather than only leaf tips or edges. For trees and shrubs, equal portions of leaves should be collected from all sides of the tree and pooled to make up the 200 gram sample.

6. An attempt should be made whenever possible to obtain plant tissue that is free of dew or other moisture.

7. Samples should not be washed or in any way treated to remove particulate materials which may have collected on the plant.

8. Plant tissue is stored in the laboratory in labeled and ventilated bags at temperatures of 2-8° C until it is dried at 80°C for 24 to 48 hours.

9. The dried plant material is ground in a semi-micro Wiley mill to pass a 40-mesh sieve.

10. Homogenous replicate samples should be saved in the event additional analyses are required.

11. Similar uncontaminated plant species to those collected from contaminated areas should be analyzed for background fluoride levels.

It should be kept in mind with plant sampling that the uptake of fluoride from the air and the subsequent distribution within the plant is not uniform. Leaf edges and tips have higher fluoride concentrations than other portions of the plants. Older leaves also often exhibit higher fluoride levels. The location of a given plant in the landscape also influences the fluoride content of the tissues. Plants on the lee side of hills, buildings or other taller vegetation often have lower fluoride levels than plants on windward locations facing a source of airborne fluorides. Note that there is no attempt to collect samples free of dust deposited as a result of raindrop splash or from dusty roads or other possible sources of fluoride. It is immaterial to the physiology of grazing animals whether fluorides are deposited as particulate on the plant surface or incorporated into the internal plant tissues.

Fluoride concentrations in the vegetation are analyzed using the semi-automated alizarin fluoride blue-lanthanum colorimetric method.

Fluoride

Fluoride is measured by the double tape sampler method described in Methods of Air Sampling and Analysis, Second Edition (Method 42222-02-72T). In this method air is drawn through a heated air inlet tube and is passed through acidic and basic treated paper tapes to remove particulate and gaseous fluorides respectively.

Indexed tapes, protected from exposure to hydrogen fluorides in the field, are sequentially passed through the air stream prior to transfer into the laboratory for analysis. Individual sample spots are cut out, treated chemically before colorimetric analyses.

Two additions to the standard procedure include the measurement of flow at the sample inlet and the utilization of a stainless steel inlet heater which prevents moisture condensation on the sample tapes.

Fluoride concentrations, expressed as hydrogen fluoride in parts-per-billion will be measured by the semi-automated alizarin fluoride blue-lanthanum colorimetric method described in Methods of Air Sampling and Analysis, Second Edition (Method 12202-02-68T).

Hydrogen Sulfide

Hydrogen sulfide is measured by the methylene blue spectrophotometric method described in Methods of Air Sampling and Analysis, Second Edition (Method P & CAM 126). In this method, hydrogen sulfide is absorbed from air bubbled through a solution of cadmium hydroxide. The hydrogen sulfide forms a suspension of cadmium sulfide which is reacted with two chemicals to produce methylene blue. The amount of methylene blue produced is measured spectrophotometrically. The measured hydrogen sulfide and the known amount of air bubbled through the cadmium hydroxide solution enables the calculation of the concentration of the hydrogen sulfide in the air. The accuracy of this method at concentrations of hydrogen sulfide below 0.8 ppb is not proven.

Lead

Lead will be measured by the atomic absorption method adopted by EPA under federal Clean Air Act (Federal Register Vol. 43, Oct. 5, 1978, pp. 46258-61). This method determines the quantity of lead in suspended particulate matter collected over 24 hours on a high volume air sampler using fiber glass filter media. The procedure involves extraction of the particulate matter using nitric acid facilitated by heat or by a mixture of nitric and hydrochloric acids facilitated by ultrasonication and determination of lead levels using an atomic absorption spectrometer with an air-acetylene flame. The measured lead on the filter and the known amount of air that passed through the filter enables the calculation of a concentration in the air in micrograms per cubic meter.

Nitrogen Dioxide

Nitrogen dioxide is measured by the gas phase chemiluminescence method adopted by EPA under the federal Clean Air Act (40 CFR Part 50, App. F). In this method, atmospheric concentrations of nitrogen dioxide are measured indirectly by measuring the light emitted from the chemiluminescent reaction of nitric oxide with ozone. Two samples of the ambient air are collected. In the first, nitrogen dioxide is reduced to nitric oxide by means of a converter. Nitric oxide, which commonly exists in ambient air together with nitrogen dioxide, passes through the converter unchanged yielding a total nitric oxide concentration equal to the sum of the concentration of nitric oxide and nitrogen dioxide, which is then measured by the chemiluminescent reaction described above. The second sample of air is measured without passing it through the converter, so only the concentration of natural nitric oxide is measured. The difference in these two measurements is the concentration of nitrogen dioxide and can be read as ppm s of nitrogen dioxide.

Ozone

Ozone is measured by the chemiluminescence method adopted by EPA under the federal Clean Air Act (40 CFR Part 50, App. D). In this method, ambient air and ethylene are delivered simultaneously to a mixing chamber where the ozone in the air reacts with the ethylene to emit light which is detected by a photomultiplier tube. The resulting photocurrent is amplified and can be read as ppm s of ozone.

Settled Particulate Matter

Settled particulate is measured by the dustfall jar method described in Methods of Air Sampling and Analysis, Second Edition (Method 21101-0170T). In this method, particulate matter is allowed to settle into a suitable wide-mouth container which is exposed to the outdoor air for 30 days. Antifreeze, algicides, and/or fungicides will have to be added to the collection jar on occasion to prevent erroneous readings. The collected material is dissolved in water and filtered through a large mesh screen to remove any bugs or other extraneous matter. The insolubles are filtered out on a paper filter and weighed. The solubles are weighed after the water is allowed to evaporate. The reported settled particulate weight is the total of the soluble and insoluble fractions.

Sulfur Dioxide

Sulfur dioxide is measured by the pararosaniline method adopted by EPA under the federal Clean Air Act (40 CFR Part 50, App. A). In this method, sulfur dioxide is absorbed from air bubbled through a solution of potassium tetrachloromercurate (TCM). A dichlorosulfitomercurate complex, which resists oxidation by the oxygen in the air, is formed. Once formed, this complex is not affected by strong oxidants (e.g. ozone, oxides of nitrogen). When the sample is returned to the lab, the complex is reacted with pararosaniline methyl sulfonic acid. The amount of sulfur dioxide absorbed will determine the absorbance of the resulting solution which is measured spectrophotometrically. The measured sulfur dioxide and the known amount of air bubbled through the TCM solution enables the calculation of the concentration of the sulfur dioxide in the air.

Suspended Particulates

Suspended particulates are measured by the high-volume (hi-vol) method adopted by EPA under the federal Clean Air Act (40 CFR Part 50, App. B). In this method, air is drawn into a covered housing and through a filter by means of a high flow rate blower (1.13 to 1.70 m^3/min.; 40 to 60 acfm). Particles within the size range of 100 to 0.1 um diameter are ordinarily collected on glass fiber filters which are then weighed. The mass concentration of suspended particulates in the ambient air is computed by dividing the weighed mass of collected particulates by the volume of air sampled.

Visibility

Visibility is measured by the integrating nephelometer method described in Methods of Air Sampling and Analysis, Second Edition (Method 11203-03-

76T). In this method, air is drawn into a chamber in the instrument. The total scattering power of the particles in the air is measured by shining a white light into the air in the chamber. A photoelectric cell measures the scattering of light by the particles in the sample. It does not measure the absorption of the light by the particulate, so if there are a significant number of black particles the measured value may overestimate the visibility. The data are reported as a measured scattering coefficient.

APPENDIX F

POLLUTANTS FOR WHICH STANDARDS WILL BE CONSIDERED LATER

As noted in Chapter III, there are several pollutants which are not included in the proposed ambient air quality standards, but which might be proposed later. This appendix discusses these pollutants.

Arsenic

Arsenic pollution in the air originates primarily from pesticide residues and from the smelting of ores containing arsenic. Until recently the Anaconda copper smelter was the largest industrial source of arsenic pollution in the United States. Ambient measurements of arsenic found a maximum 24-hour average of 1.8 ug/m^3 in Anaconda and .25 ug/m^3 in Butte and a two-month average of .32 ug/m^3 in Anaconda and .05 ug/m^3 in Butte during March and April of 1978.

Arsenic compounds are believed to be a cause of lung cancer, possibly acting in conjunction with sulfur dioxide (Lee and Fraumeni, 1969; Ott _et_ _al_., 1974; Matanoski, 1976). A significantly increased incidence of lung cancer has been reported among residents living near the Anaconda Copper Smelter (Newman _et_ _al_., 1975). An occupational exposure standard of 10 ug/m^3, averaged over eight hours, has recently been adopted by the Occupational Safety and Health Administration (OSHA, 1978).

Current research has not identified a safe level of community exposure to arsenic compounds. Concentrations permitted for occupational exposure would not be appropriate for a standard designed to protect the

public outside the work place. The Air Quality Bureau proposes to continue to actively review the scientific information on arsenic and to propose an ambient air quality standard when adequate information is available.

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Beryllium

Beryllium was refined in Anaconda until 1971. No plans to resume production are known. Wastes stored nearby may be a future source of beryllium exposure.

Inhalation of low concentrations of beryllium can cause lung injury and death (Hardy and Stoeckle, 1959). In one community, exposure to ambient beryllium levels of .015 ug/m^3 resulted in 26 cases of beryllium poisoning (Sussman, 1959). Beryllium is known to cause lung cancer in animals (Schepers, 1961) and is suspected as a cause of lung cancer in humans (Waggoner, 1978).

The Nuclear Regulatory Commission and the Environmental Protection Agency have adopted a community exposure standard of .01 ug/m^3, 30-day average (EPA, 1973).

There is no present significant source of beryllium in Montana, so there is no immediate need to propose an ambient standard. New information on the cancer-causing properties of beryllium and the safe level of community exposure are expected to be available in the near future. The Air Quality Bureau proposes to continue to actively review the scientific information on beryllium and to propose an ambient air quality standard when adequate information is available.

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Cadmium

Cadmium pollution enters the air primarily from smelters that process ores containing cadmium. The Anaconda copper smelter and the ASARCO lead smelter and zinc plant in East Helena are major sources of cadmium pollution. In 1971, 24-hour average concentrations of cadmium were measured in East Helena from .06 to .29 ug/m^3. In March and April, 1978, a maximum 24-hour average concentration of .12 ug/m^3 was measured in Anaconda.

Exposure to cadmium can result in irreversible damage to the kidneys (Kazantzis *et al*., 1963; Kjellstrom *et al*., 1977). Softening of the bone and spontaneous fractures have been observed in some persons exposed to ambient cadmium pollution (Emerson, 1970). Cadmium has been shown to cause cancer in animals (Roe *et al*., 1964) and is strongly suspected as a cause of cancer in humans, particularly prostate cancer (Lemen *et al*., 1976).

An analysis of total cadmium exposure in East Helena estimated that .013 ug/m^3 cadmium in the air was low enough to avoid causing kidney damage (Rupp *et al*., 1978). This level would not necessarily protect the public from other health effects. The Air Quality Bureau proposes to continue to actively review the scientific information on cadmium and to propose an ambient air quality standard when adequate information is available.

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Odors

State regulatory agencies currently use two methods to control odorous air pollution. Seventeen states simply prohibit any odor that creates a public nuisance. It is the responsibility of the regulatory agency to determine if a particular smell is, in fact, a public nuisance. No objective measure is set to establish this. Six states have established a percentage of the surrounding population that must be affected before any action is taken (e.g., 30 percent of the people interviewed must say that the odor is objectionable) (Leonardos, 1964).

An alternative approach is based on the concentration at which an odor becomes perceivable. Many odorous compounds can be smelled at concentrations too low to be easily measured, so the regulations are written in terms of how much clean air must be mixed with the odorous air to achieve an odor concentration too weak to be detected by half the members of an odor panel. Several methods are available for measuring odors in this way. The most reliable is the dynamic forced-choice triangle olfactometer (Dravnieks et al., 1978).

Huey et al. (1960) concluded that odors more than seven times the threshhold level will cause complaints to the authorities. Odors stronger than 31 times threshold were deemed a serious public nuisance. Six states and the District of Columbia have adopted an ambient air quality standard for odors based on these principles. Two states permit higher concentration in industrial areas (Leonardos, 1974). A hydrogen sulfide standard used by many states (.03 ppm) is approximately seven times the average detection threshold (.0045 ppm).

Very little analytical work has been done on the response of the community to varying levels or types of odors. Lindvald (1970) found that a subjective evaluation of odor intensity increased steeply (logarithmically) when odorant concentrations increased. Jonsson et al. (1975) measured the community response to pulp mill odors and Deane and Sanders (1978) measured community reaction to petroleum refinery odors. Jonsson et al. found 31 percent of the community bothered by the odor in an area where the odor was three times the odor threshold and 50 percent bothered in an area where the odor was about eight times threshold. No other studies have been published which could support or modify the observations of Huey et al.

Measurements for hydrogen sulfide are taken routinely in Missoula. These are summarized in Table XII in Appendix A. Measurements have also been made for highly odorous methyl mercaptans in Missoula. The high 30-minute average value for the observation period ranged from 10 to 60 times threshold (DHES, 1974).

The Air Quality Bureau presently has a public nuisance standard for odors. Such standards have been used effectively in the past and do not require changing at this time.

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Polycyclic Organic Matter

Polluted air contains various organic compounds, including the hydrocarbons involved in the photochemical production of ozone. The organic compounds causing some of the greatest concern are those containing two or more benzene rings in their molecular structure (thus, polycyclic), including polynuclear aromatic hydrocarbons (such as benzo(a)pyrene), various arenes, the polychloro compounds, and some pesticides. Polycyclic organic matter (POM) is expected to be found as a vapor or as a condensed coating on the surface of fine particles and to a much lesser degree on the surface of coarse particles. The primary sources of POM's are wood-fired residential fireplaces, oil-and coal-fired furnaces and boilers, and motocycles and diesel-powered automobiles. Aluminum reduction plants are also a source of POM. No recent measurements of POM have been made in Montana.

Benzo(a)pyrene has been identified as the principal cancer-causing agent in soot. About two dozen other polycyclic organic compounds also have been shown to cause cancer in animals. The presence of inert respirable particulate enhances the cancer-causing effect in the lungs (Saffiotti *et al.*, 1972). POM's have also been shown to be involved in the formation of breast cancers (Huggins, 1965). POM injures the bone marrow and destroys the ability to form blood cells (Cawein and Syduor, 1968). Studies of workers exposed to coke oven emissions, which are high in benzo(a)pyrene, have clearly established a much higher risk of lung and kidney cancer and a higher incidence of other respiratory diseases among these workers (Redmond *et al.*, 1976).

Excess deaths due to lung cancer were found in communities with petroleum refineries, as compared to otherwise similar cities (Blot et al., 1977). Excess deaths due to lung cancer were specifically related to measured POM levels in Los Angeles (Henderson et al., 1975). A British study found a strong correlation between the incidence of lung cancer, the amount of coal burned, and POM concentrations (Stocks, 1966, 1967).

No information is available on community exposures to polycyclic organic matter in Montana or on the levels which would protect the public health. Therefore, the Air Quality Bureau proposes to continue to review the scientific information on POM and to conduct measurements to determine the ambient concentrations. The Air Quality Bureau intends to propose an ambient standard when adequate information is available.

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Reactive Sulfur (Sulfation)

Most ambient air quality standards regulate concentrations of a specific chemical species. Standards governing reactive sulfur (sulfation), on the other hand, control any airborne sulfur compound that can react with a lead dioxide collector. Sulfur dioxide, hydrogen sulfide, sulfite, methyl mercaptans, and sulfurous and sulfuric acids are all measured and recorded by this technique.

The reactive sulfur or "lead candle" method, as it has been called, was originally developed as a cheap, reliable measuring technique for sulfur dioxide. At a single location, during a single weather season, reasonably good correlations are possible between results from this technique and other more specific measurement methods (Lynch *et al.*, 1978). However, substantial errors can be caused by inconsistent weather, variations in preparation of collector plates, and interference by other pollutants (McAdie and Jones, 1971). Pollution control officers in states with reactive sulfur standards recognize the inaccuracies encountered when attempting to use the method to measure sulfur dioxide. For this reason most agencies define "reactive sulfur" as "that which is measured by sulfation."

It has been suggested that the sulfation air quality standard should be retained because various health and welfare effects have been observed at high sulfation levels. While this is true, a level of sulfaction caused by a certain mix of pollutants and weather in one area does not necessarily have the same health and welfare implications as a similar sulfation level from a different pollutant mix and weather in another area. This non-specificity of the measurement makes it impossible to compare research studies to develop a standard.

In Montana, there are a number of airsheds with different weather conditions and different sulfur compound mixes, some more harmful than others. Because of the uncertainty in applying one sulfation standard to the entire state throughout the year, the Air Quality Bureau will not propose a reactive sulfur (sulfation) standard. Further, health and environmental effects information is not available to allow proposal of specific sulfation standards for designated portions of the state.

Reactive sulfur nevertheless remains a useful tool for extending an ambient monitoring network to characterize the general air quality of an airshed. As a planning tool, sulfation provides a low cost method of determining where additional and more accurate monitoring techniques should be used. The Air Quality Bureau will continue to use reactive sulfur in this capacity in the future.

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Respirable Particles

As described in "Sulfur Oxides and Particulate" inahled particles above a certain size are filtered out by the nose. Smaller particles remain in the air stream and eventually reach the lungs. If a particle is deposited in the lungs it is much more likely to result in an adverse health effect than if it is swallowed and passes through the stomach. For this reason many people have advocated establishing an ambient air quality standard for such "respirable" particles (Perera and Ahmed, 1978).

Several attempts have been made to define a size range of particles that may be associated with adverse health effects. Some have suggested that standards should be set for particles below 2.5 micrometers. Others, observing that a substantial fraction of the larger particles enter the lung, have designated various particle sizes between 3.5 and 9 micrometers as "respirable." Recently, the Environmental Protection Agency has proposed identifying particles less than 15 micrometers as "inhalable," recognizing that even particles that do not reach the deep lung can have a significant adverse health effect in the nose, throat, windpipe, or digestive tract. Currently, no single definition of "respirable" particles is accepted by all researchers.

Particles smaller than 2.5 micrometers are formed primarily from combustion or the chemical conversion of gases in the atmosphere. Sulfur dioxide, for example, converts to sulfate particles. As much as two-thirds of the fine particulate may be sulfates (Sevens et al., 1978). In California organic compounds (which often include some of the cancer-causing polycyclic organics) were found to be the dominant constituent of fine particulate (Hidy, 1975). Fine carbon particles (perhaps from motor vehicle exhaust) also have been identified as a major constituent of the fine particulate (Rosen and Novakov, 1978). These particles may be particularly active absorbers of sulfur dioxide and other gases, providing a "carrier" to take these pollutants deep into the lung. Inhaled fine particulate may also interfere with the foreign matter removal mechanisms in the lung and prolong the contact between pollutants and lung tissue (Saffiotti, 1972).

A few epidemiological studies have measured the effects of respirable particulates. Cohen et al.(1972) and Kenline (1966), in studies discussed in "Sulfur Oxides and Particulate" found asthma attack rates increased at higher respirable particulate levels. Stebbings and Hayes (1976) observed a correlation between increased respirable particulate levels and increased aggravation of heart and lung disease symptoms among elderly persons. Hammer (1977) and Chapman et al. (1976) in the studies discussed in "Sulfur Oxides and Particulate" found increased incidence of illness and decreased lung function in children in Birmingham where the average levels of respirable particulate were higher. Unfortnately, no consistent measurement technique was used nor was the definition of "respirable particulate" the same in these studies.

With the data now available it is not possible to identify a threshold of size range or concentration of respirable particulate that would protect the public health to a greater degree than a standard for total suspended particulate. Therefore the Air Quality Bureau proposes to continue reviewing the scientific information on respirable particulates and to conduct size-specific measurements of suspended particulates to determine the actual concentrations in Montana. The Air Quality Bureau will propose a standard for respirable particulate when adequate information is available.

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Suspended Sulfate and Sulfuric Acid Mist

There have been repeated suggestions that the observed adverse health effects of sulfur dioxide are actually caused by suspended sulfates, particularly sulfuric acid. Although chemically incorrect, the term "sulfates" is used to refer to sulfites and sulfurous acid, both of which have been suspected to cause health effects.

Some sulfate is emitted from sources directly, but most is formed in the atmosphere by the oxidation of sulfur dioxide, as discussed in "Sulfur Oxides and Particulate." All such "secondary" particles are small enough to be breathed into the deep lung.

Clinical studies discussed in "Sulfur Oxides and Particulates" demonstrate the adverse effects of sulfates on animals (Amdur, 1978; Fairchild, 1975) and people (Bell et al., 1977; Toyama and Nakamura, 1964). Some epidemiological studies discussed in "Sulfur Oxides and Particulates" correlated increased sulfate levels with decreased lung function (Chapman et al., 1976; Shy et al., 1973) and increased illness (Hammer, 1978, Cohen et al., 1972, Lawther, 1970).

Four studies conducted by the Community Health and Environmental Surveillance System (CHESS) program of the EPA attempted to quantify the critical exposure levels for adverse health effects from suspended sulfate (Finklea, et al., 1974). Summarizing these studies, the EPA concluded that aggravation of existing conditions could be observed when daily average sulfate levels rose above the 6 to 10 ug/m^3 range. Increased incidence of respiratory disease was observed when annual average sulfate levels were above 9 to 15 ug/m^3 (EPA, 1975). However, these studies have come under severe attack because of problems associated with program management, questionable analysis of some ambient samples, arbitrary assumptions forced onto some data and disagreement over the design of some parts of some of the studies (Committee on Science and Technology, 1976).

The Air Quality Bureau has set aside the CHESS results because of the controversy surrounding them. Without the CHESS studies only limited information is available as to the sulfate levels that are safe for humans.

The effects of sulfates on plants and the environment result primarily from increased acidity in precipitation. Acid rain is discussed in "Sulfur Oxides and Particulates." The specific effects that may occur in various parts of Montana depend on the characteristics of the individual soils and plants and the amount and frequency of the acid rain. Soils in the Bitterroot mountains in western Montana are known to be particularly sensitive to acid precipitation. Other soils in Montana also may be sensitive (Galloway and Cowling, 1978). Techniques for measuring the acidity of rainfall are well established (Brosset, 1976; Galloway and Likens, 1976).

Measurement of sulfate in the ambient air is more difficult. Traditionally the particulate collected in a high volume filter was analyzed for sulfate by wet chemistry techniques. However, there is considerable evidence that such measurements err substantially due to the conversion of sulfur dioxide gas to sulfate on the filter. This conversion can be minimized by pre-treatment of the filter blanks, but previous measurements of sulfate are seriously in question. Other techniques for measuring sulfates are currently being developed, with some of these techniques capable of determining specific chemical species. Currently, none of these techniques can be used for ambient measurements under field conditions (Newman, 1978).

Although the data suggests that an acidity level (pH) of 5.4 in precipitation is weak enough to be safe for the environment, it is not clear whether such a standard is necessary. The Air Quality Bureau proposes to continue to review the scientific information on sulfates and acid precipitation and to measure precipitation acidity in Montana. When such efforts are conclusive, the Air Quality Bureau will propose a suitable standard for acid precipitation.

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